

# JOURNAL of the American Veterinary Medical Association

FORMERLY  
AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

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The American Veterinary Medical Association

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# JOURNAL

## OF THE

### American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 735 Book Building, Detroit, Mich.

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## PUBLIC SENTIMENT WITH THE VETERINARIANS

The existence of foot-and-mouth disease in California, for the three months last past, has been the subject of much comment in the newspapers throughout the country. As would be only natural to expect, many of the press notices which have appeared have carried erroneous or ridiculous statements, but on the whole, when compared with the authoritative reports made by federal and state officials, it is safe to say that the seriousness of the situation has been in no way exaggerated.

The following editorial appeared in *The Globe-Gazette*, Mason City, Iowa. It is an unusually well written endorsement of the drastic measures which it has been necessary to adopt in an effort to eradicate the infection:

"Those acquainted with the ravages of hoof-and-mouth disease may be disposed to point a finger of censure at the drastic means employed by Pacific coast states to stop the disease. Those who have seen the disease in its awful manifestations will take the position that no methods could be too drastic.

"Within the past week hundreds of tourists have been held up at the Arizona state border as a quarantine regulation. Tales of suffering and the like, mostly unverified, have come from the scene of the enforced waits and one or two groups of the stranded tourists have made a dash through quarantine.

"The ordinary Iowa farmer would recommend a heavy penalty for these persons. While these tourists have a right to eat and live, they have no right to violate the rules which have been decided on as essential,

if the spread of the livestock maladies is to be halted. Arizona farmers probably have a stronger case against these wilful travelers, but Iowa farmers are righteously concerned, for the reason that Arizona is closer to Iowa than California.

"Authorities charged with stopping the spread of one of the most highly communicable diseases known to animal life are being forced to meet an emergency. Their methods are probably not the same as they would be if the campaign could be studied out in advance. The unmistakable thoroughness with which they are attacking the problem, however, is more than making up for any impolite abruptness of which they may be guilty."

The Ludington (Mich.) *News* apparently appreciates the seriousness of the situation. Having in mind the fact that on two previous occasions Michigan has been visited with the plague, the following editorial comment recently appeared:

"Arizona's blockade against tourist motor cars from California may rouse some national interest in the Pacific coast wrestle with that most dangerous and destructive of live stock plagues, apthous fever, or foot-and-mouth disease.

"It is a virulent, highly infectious disease, spreading by contact to most farm animals and to man. In the Middle West, in 1908, it all but wiped out the cattle and sheep industries in many counties. The one and only sure way of stamping it out is to destroy the animals in the infected zone, to bury the carcasses in quicklime and to enforce a quarantine so rigid that nothing may pass.

"Virtually every state on the west coast and in the mountain areas has barred live stock and certain other shipments from California. The range country is in a panic as this apthous fever climbs the western slopes of the Cordilleras. Infection may be carried on the clothing, the boots, vehicle wheels, on anything to which it may cling. Birds and dogs may carry it. Anything moving from an infected to a clean area is dangerous.

"That is why Governor Hunt, of the great cattle state of Arizona, is taking drastic measures and why the motorist is being held up and fumigated or turned back at the California line. That also is why President Coolidge is calling the governors of ten western states and of Hawaii to a conference for framing new quarantine resolutions. Once over the Rockies this plague may sweep farm and range. To the harassed tourist the whole business may seem an outrage, but to the live stock country that knows what an outbreak means, it is a serious situation."

It is pleasing to note that the country generally has endorsed every move to control and eradicate the disease. Twenty-two states can sympathize with California, having been through it just a few years ago. The memory is fresh. Like the burnt child who avoids the fire, they want the disease kept at a distance, even California is too close for comfort. The Fort Madison, Iowa, *Democrat* takes things very philosophically, when it says:

"It is pretty hard for persons unacquainted with the foot-and-mouth disease and the situation that confronts the western coast to appreciate the desperate conditions there. We fall into ready sympathy with the tourists who have been held up on account of the quarantine and are likely to think that such rigorous measures are unnecessary, but when one realizes the havoc that the terrible disease is capable of and the menace that an epidemic presents, it may lead to a better understanding.

"Already there have been slain by the state and federal officials more than fifty thousand head of stock—cattle, sheep and hogs. They were

not merely killed but were buried in quicklime at a depth of five feet. The federal congress, without even a suspicion of opposition, had appropriated a million and a half dollars to eradicate the disease, and the situation grows graver as fresh outbreaks occur.

"Unless the epidemic is checked and that quickly there is danger that the disease might sweep the whole country and that domestic animals throughout the breadth of the land fall victims to it with a financial loss to the individuals that hardly can be computed. It would mean that the whole country would be infected and that until every trace of the disease were stamped out, there could not be any resumption of live stock raising without the greatest risk.

"The whole public would suffer for years in the inevitable high prices of meat, and there likely would ensue for years a near famine in beef and pork and mutton. So that it is not only California and the bordering states that are interested in the desperate fight that is being made now, but every person who owns animals or who eats meat. And if the meat situation grows tense and the supply is diminished, there would be a sympathetic effect on other foods and the vegetarians who may be contemplating the situation with smugness, would find that they too, were affected by the plague.

"In these days of a high state of civilization, we do not stand apart from our neighbors. When they suffer, we suffer. Hardly a thing can happen in any part of the globe without its effects reaching to the remotest hamlet. And California is not alone in her foot-and-mouth disease. The rest of us are sitting hard by, waiting with a keen interest the successful outcome of the fight on the epidemic."

A little inside information is gained from a report which has been given wide publicity in the press:

"Nearly every industry in California is affected by the rapid spread of foot-and-mouth disease among cattle in this State.

"All state products are under embargo and financial losses will mount into the millions. Tourists are stranded in the state, unable to leave. Cattle are being slaughtered by the thousand. As a result, the state is calling on the Federal Government to rescue it from its desperate situation.

"The disease started in live stock herds early in the year. Ordinarily it could have been easily checked by inspectors of the state Agricultural Department. However, that Department had been reduced in size and its appropriation cut as part of an "economy" program, and it was unable to cope with the situation."

From Los Angeles, *The Times* comments on the shortage of veterinarians available to fight the disease. We find it difficult to reconcile this report with the information given in the JOURNAL last month, to the effect that California has a surplus of veterinarians. *The Times* says:

"The State-wide fight to control the foot-and-mouth disease in California has brought to light the fact that there are all too few veterinarians in the country to meet the ordinary requirements of the live stock industry, let alone to effectively handle an emergency situation such as that presented by the sudden outbreak of a dangerous epidemic among animals. The State, Federal and county authorities have been handicapped in their efforts to combat the scourge now confronting the stock raisers of California by the difficulty in recruiting trained veterinarians, and it has become evident that any other state would find itself in about the same predicament in the face of a like menace. . . .

"It may be that one of the officials on the firing line in the California hoof-and-mouth fight was right when he said last week that 'we need cow doctors and not so many family physicians.' "

Governor Richardson, of California, has been freely quoted in the press, in connection with the outbreak of foot-and-mouth disease in the Golden State. One of his recent statements, in commenting on an appeal for troops by the Madera County sheriff, is as follows:

"Veterinarians, not soldiers, are what California needs in its fight to eradicate the foot-and-mouth disease.

"More veterinarians and less hysteria is the State's first requirements just now. Troops can't eradicate the foot-and-mouth disease in Madera county, and so far as I'm concerned no troops will be sent. What is more, the federal government is now in charge of the situation."



Iowa State Capitol, Des Moines

### SIoux CITY'S 1924 CAMPAIGN

Encouraged by the results of their 1923 publicity campaign, the officers of the Sioux City Serum Company have decided to put on a similar campaign this year. It will be recalled that their 1923 advertisements all stressed the desirability of farmers using veterinary services. The same thought runs through the 1924 series, proofs of which it has been our privilege to examine.

"Have a veterinarian vaccinate your pigs," says the first of the series. "Pigs should be vaccinated . . . . . by a veterinarian," says the second. "Always have a veterinarian do the work," advises the third, and so on through the series. One advertisement carries the interesting statement that "hog cholera losses (last year) would have purchased 97,977 Fords."

President Cusack states that the "Sioux Brand" campaign started May 15, and space has been engaged in eight farm papers and six swine journals. It is estimated that those who will read these advertisements will number several millions. That ought to help the cause some.

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### THE BUREAU HAS A BIRTHDAY

The Bureau of Animal Industry was forty years old on May 29, 1924. Created in 1884, by an act of Congress, for the purpose of coping with contagious pleuro-pneumonia, the fortieth anniversary of the birth of the Bureau found it engaged in a death-grapple with one of the most insidious foes that it is ever called upon to fight, foot-and-mouth disease.

During the forty years of important public service, first under the brilliant leadership of Dr. Daniel Elmer Salmon (1884-1905), then under his worthy successor, Dr. Alonzo Dorus Melvin (1905-1917), and since 1917 with that master pilot, Dr. John Robbins Mohler, at the helm, the Bureau has time and time again demonstrated its great worth, and now with a personnel numbering in excess of five thousand employes, approximately one-third of whom are veterinarians, it is regarded as the greatest animal-disease-fighting machine in the world.

The average citizen has no conception of the ramifications of the Bureau's activities. Protecting, as it does, an industry valued at ten billion dollars, largely through its efforts the United States has been made the safest country in the world for the investment of capital in live stock. Human health is directly benefited, to an incalculable degree, through the agency of the meat inspection service which safeguards two pounds of meat out of every three consumed in this country, not to mention other lines of work done with diseases communicable to man.

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### AN OPPORTUNITY TO SERVE?

The army veterinarians have been getting a great deal of publicity lately. Newspaper reports have told about them treating the army horses with chlorine gas to cure influenza. We have also read about the army mules being subjected to "vacuum cleaning." But we have not seen anything about veterinarians around Washington being consulted as to the best method for removing oil stains from a certain famous elephant and an equally well-known donkey.

### EXECUTIVE BOARD ELECTION

The polls closed on May 2, for the election of a member of the Executive Board for District Number 4. The election board, consisting of Dr. S. Brenton, Dr. E. P. Schaffter and the Secretary-Editor, canvassed the ballots and found that Dr. John R. Mohler, Washington, D. C., had the highest number of votes and declared Dr. Mohler elected, for a term of five years, beginning with the close of the 1924 meeting, in Des Moines.

Any introduction of Dr. Mohler to our members would be superfluous. He was a member of the first Executive Board



DR. JOHN R. MOHLER

elected under the present constitution, and represented what was then District Number 3, now Number 4. He was a member of the Subcommittee on Journal, from 1917 until 1920, when he was appointed Editor, succeeding Dr. W. H. Dalrymple, and serving in this capacity until 1923. Dr. Mohler served the A. V. M. A. as president, 1912-1913.

With the election of Dr. Mohler to the Executive Board, this body will, for the second time, include in its membership the ranking veterinary sanitary control officials of both the United States and Canada.

## COMING VETERINARY MEETINGS

- New York State Veterinary Medical Society. (Joint meeting with Eastern States Tuberculosis Eradication Conference.) Albany, N. Y. June 10-11-12, 1924. Dr. C. E. Hayden, 110 Irving Place, Ithaca, N. Y.
- Arkansas Veterinary Association. Marion Hotel, Little Rock, Ark. June 21, 1924. Dr. Joe H. Bux, Secretary, Old State House, Little Rock, Ark.
- Michigan State Veterinary Medical Association. East Lansing, Mich. June 24-25-26, 1924. Dr. E. K. Sales, Secretary, East Lansing, Mich.
- North Carolina State Veterinary Medical Association. Blowing Rock, N. C. June 25, 1924. Dr. J. P. Spoon, Secretary, Burlington, N. C.
- Missouri Veterinary Medical Association. Excelsior Springs, Mo. June 25-26, 1924. Dr. Fred C. Cater, Secretary, Sedalia, Mo.
- Texas State Veterinary Medical Association. Brownwood, Texas. June 25-26, 1924. Dr. F. S. Palmer, Acting Secretary, 3018 Commerce St., Dallas, Texas.
- Massachusetts Veterinary Association. American House, Boston, Mass. June 25, 1924. Dr. C. H. Playdon, Secretary, Reading, Mass.
- Illinois State Veterinary Medical Association. St. Nicholas Hotel, Springfield, Ill. July 9-10, 1924. Dr. L. A. Merillat, Secretary, 1827 So. Wabash Ave., Chicago, Ill.
- Kentucky Veterinary Medical Association. Frankfort, Ky. July 9-10, 1924. Dr. J. A. Winkler, Secretary, Newport, Ky.
- Virginia State Veterinary Medical Association. Ocean View Hotel, Ocean View, Va. July 10-11, 1924. Dr. H. T. Farmer, Secretary, 316 N. Henry St., Richmond, Va.
- New Jersey Veterinary Medical Association of. Asbury Park, N. J. July 10-11, 1924. Dr. P. B. Silvester, Secretary, Princeton, N. J.
- Oklahoma State Veterinary Medical Association. Medicine Park, Okla. July 14-15-16, 1924. Dr. L. B. Barber, Secretary, 100 Live Stock Exchange, Oklahoma City, Okla.
- Maryland State Veterinary Medical Association. College Park, Md. July 17-18, 1924. Dr. E. M. Pickens, Secretary, College Park, Md.

## THE PATHOLOGY OF POSTERIOR PARALYSIS

By S. A. GOLDBERG

*Department of Pathology and Bacteriology*

*In cooperation with*

L. A. MAYNARD, K. V. WILLIAMS, and O. B. CHRISTY

*Department of Animal Husbandry, Cornell University*

*Ithaca, N. Y.*

### INTRODUCTION

In the course of winter feeding of pigs, it has often been observed that they will become stiff and fail to make normal gains. To this condition such terms as "rheumatism," "pig-gout," "rickets," "scurvy," "leg-weakness," and "posterior paralysis" have been applied.

Plimmer<sup>1</sup> observed four young pigs that became ill on a diet of cooked food. The symptoms suggested scurvy. They were:

- (1) Stoppage of growth.
- (2) On being approached the animals squealed with more than their usual fervor, and on being touched the squeal became frenzied.
- (3) Lack of appetite and a condition of great lassitude.
- (4) Spasmodic twitching of limb muscles.
- (5) Swollen joints.
- (6) Inability to walk or stand properly. On being made to rise or move, the pigs moved about with the trotters bent underneath the forelimbs. The hind limbs were also feeble and not held straight as in normal pigs, but the trotters were not bent under as in the forelegs."

Plimmer concluded that it was not rickets, since the anti-rachitic factor is fairly stable to heat, while the antiscorbutic factor is not. Turnips (Yellow Swede), given fresh from the field as a therapeutic, cured one in fourteen days. The others, after four weeks, gradually regained their strength. Histological examination of bone lesions is lacking in this paper. The gross lesions of one pig were reported. They are as follows:

"The ribs on one side appeared normal; on the other side one rib showed a healed fracture (of old standing); and five other ribs showed hemorrhage. Two of these ribs were curved.

"The bones of one side looked normal, on the other showed thickening of the radius and ulna, and fusion and thickening of the tibia and fibula."

Zilva et al.,<sup>2</sup> in an experiment on two pigs from birth on, in which the fat-soluble factor was restricted in the diet, and two others that were fed the vitamin in abundance, observed leg weakness in the two that received an abundant amount of the vitamin as well as those that did not. The pathology of the ribs of one in which the diet was deficient in the vitamin and the

two that received a large supply of it, is as follows:

"(1) A moderate increase in the number of proliferating and hypertrophic cartilage cells was found in the rib of the pig in which the diet was lacking in vitamin.

"(2) A deep penetration of blood vessels from the bone marrow into the cartilage was observed in all the three cases.

"(3) The presence of fibro-cellular tissue instead of normal marrow was found in all cases. It was most pronounced in the animal where the diet was deficient in the factor."

Zilva concludes that "when pigs are 'off their feet' it does not necessarily imply that it is due to rickets."

In a feeding trial conducted by the Animal Husbandry Department, in the fall of 1921, symptoms similar to those described by Plimmer were observed in two pigs that were fed the following diet:

Yellow hominy	2 parts
Standard wheat middlings	1 part
Skim milk	1 part

Where barley was used instead of hominy no trouble resulted. No histological examination of bones was made. The pigs were turned out and soon recovered.

The experiment was repeated the following summer, but the pigs remained normal. This can perhaps be explained on the ground that summer milk is richer in the vitamin factor than winter milk.

Golding, Zilva, Drummond and Coward<sup>3</sup> conducted a very careful experiment on "The Relation of the Fat-Soluble Factor to Rickets and Growth in Pigs." A sow on a diet of toppings (middlings) and whey, lacking in vitamin A, manifested deprivation by retarded growth. With the addition of Lucerne, the animal resumed growth. After 74 days of normal growth she was bred by a pure-bred Berkshire boar, and again placed on a diet of toppings, whey and swedes. This diet was so planned that the young would not be born with a supply of the vitamin.

After 116 days, 9 pigs were born, and at the end of 65 hours, 8 were divided into two sections. One section received vitamin A from the mother, and the other received an addition in the form of cream ( $\frac{1}{8}$  ounce a day), which was later changed to cod-liver oil ( $\frac{1}{2}$  raising to 1 ounce a day). Two pigs in the lot receiving mother's milk as the sole source of the vitamin received a supply of inactive olive oil to balance the nutrients of the other lot receiving the cod-liver oil.

At the end of 53 days the pigs were weaned and weighed. No difference in weight between the two lots was evident. This

confirmed the earlier work of the authors<sup>2</sup> that the requirements of the pig for the fat-soluble factor are not of a high order.

The pigs were then divided into four lots, one lot (previously fed only mother's milk) was fed a diet lacking in vitamin A and calcium, another (from the same previous lot) was fed a diet deficient in only vitamin A, lot three received vitamin A, but lacked calcium, and lot four received an adequate supply of both vitamin A and calcium. The vitamin was supplied in the form of cod-liver oil. Vitamins B and C were given to all in the form of yeast extract and lemon juice.

At the end of 54 days marked differences in the lots could be seen. Group I ( $-A-Ca$ ) showed a scurvy skin, saddle back, weak legs, joints painful to pressure, and had failed to make normal gains. Group II ( $-A+Ca$ ) showed a lack of vitality and a saddle back. Group III ( $+A-Ca$ ) was in good condition. Group IV ( $+A+Ca$ ) was in the best condition.

At the end of 111 days the pigs in groups I and II were down in the hind legs. Group III showed rough skin, lack of size and bloom, but were otherwise normal. Group IV was perfectly normal.

The pigs were then autopsied and the bone lesions reported by Prof. V. Korenchevsky. No lesions were found in any of the organs. The bone lesions are as follows:

"(1) Group IV ( $+A+Ca$ ) showed a somewhat abnormal picture with very slight osteoporosis and a belated deposition of lime salts in the newly formed bone.

"(2) In all cases the bone marrow, especially in the region of secondary spongiosa, consisted of a fine fibrinous reticulum with but few bone marrow cells.

"(3) The histological examination in animals belonging to groups II and III ( $-A+Ca$  and  $+A-Ca$ ) was different from that of group IV. Only a high degree of osteoporosis could be seen.

"(4) In group I ( $-A-Ca$ ) the condition of osteoporosis was more marked. Moreover a more frequent incursion of the proliferating cartilage into the bone marrow was in evidence. In these places was also noticed defective calcification in the zone of provisional calcification. It is quite evident, in spite of the very marked changes which have been effected by our restricted diets, no rickets in the pathological sense of the word have been induced."

The report goes on to say that "the animals in groups I and II have on various occasions during the experiment displayed a condition which would have been described by the practical man as the pigs 'being off their feet.' No doubt such a condition has been, before now, loosely referred to as rickets. Although defective calcification in the zone of provisional calcification, in the case of group I, no increase in osteoid tissue could be estab-

lished, and therefore no faulty deposition of calcium in the newly formed bone in the sense of rickets can be asserted."

In the light of more recent investigation on the etiology of rickets, the pathology of the bone has a very important consideration. In endeavoring to determine the factors involved and their relative importance, much has been studied concerning the bone changes. Korenchevsky<sup>4</sup> has indicated the following points in the diagnosis of rickets:

"(1) Diminution of the calcium content of the bone.

"(2) Presence of osteoid tissue in amounts distinctly exceeding the normal.

"(3) Enlargement and disorganization of the zone of proliferating cartilage.

"(4) Absence or defect of deposition of lime salts in the zone of provisional calcification.

"(5) Absence or marked deficiency of calcareous depositions in callus after spontaneous fractures of bones. The presence of osteoporosis in the conditions named above does not prevent the diagnosis of rickets."

Maass<sup>12</sup> gives the following description of rickets. The organic disturbances of the bones produce a mechanical effect upon the growth of bone, which results in a compensatory growth in areas free from pressure; while there is a thinning and bending on the concave side (hence the rosary of the ribs). Deficient calcification leads to softening of the bony trabeculae, so that there is a mechanical irregularity of the zone of provisional calcification. He concludes that the deformities are due to a mechanical effect of the deficient calcification of the bones.

McCollum<sup>5</sup> describes the abnormal changes in rachitic bone as follows:

"In rachitic bone all the processes of growth and the maintenance of equilibrium are abnormal. Calcium salts are not deposited in the normal way. In the normal bone the junction of the cartilage with the shaft forms a straight, regular line. In the rachitic bone it is ragged because there is no provisional zone of calcification, and the cartilage is invaded by large tufts of blood vessels from the shaft, which destroy it irregularly. At the same time masses of cartilage persist where they should be destroyed, and islands remain cut off from the main body of the tissue. Osteoid tissue is not calcified, as it is formed either by the trabeculae or by the periosteum, so that the bone becomes soft. The animal attempts unsuccessfully to compensate for the weakness due to lack of lime salts by producing osteoid tissue in abnormal amounts, especially at points subjected to stress and strain (the curved side of the bone and at the insertions of muscle and tendons). This over-production of osteoid tissue and cartilage causes irregular enlargement of the bones and this weakness consequent upon the lack of lime salts results in bowing and fracture. Because of the abnormal growth at the end of the shaft, a zone known as the rachitic metaphysis forms, which consists of blood vessels, connective tissue, osteoid, marrow elements, and cartilage cells in all stages of degeneration and change into other tissue. The osteoid tissue formed shows little sign of being absorbed in rickets as it is commonly seen. The balance of forces has swung in the direction of apposition and persistence of cartilage."

Shipley<sup>8</sup> summed up the etiology of rickets in the following manner:

"Rickets is a disease of metabolism usually due to faulty food. It may be produced in rats by certain diets containing an improper balance between

calcium and phosphorus when an uncharacterized substance associated with certain fats is absent or deficiently supplied. There are two sorts of rickets in rats, one a low-calcium type, produced by diets relatively low in calcium, but containing an approximately normal amount of phosphorus, other things being equal, and second, a low-phosphate type, produced by diets deficient in phosphorus, with a normal or high calcium content. Some uncharacterized organic substance, which is present in abundance in cod-liver oil, enables the organism to compensate for a defective calcium-phosphate ratio in the food. Exposure to sunlight or to the rays of the mercury vapor quartz lamp, or to certain metallic arcs will do the same thing. Either of these factors will induce healing in the bones of rachitic animals. Starvation also will induce healing.

"These investigations so far leave a great many questions still unsettled. Some of these, besides those discussed, we are in a position to answer; others await further study for their elucidation. We cannot tell, for example, whether the gastro-intestinal tract receives these diets which we put into it and passes them on to the body without changing their composition. Nor are we now in a position to say whether or not the diets influence the body tissues directly or through some unknown mechanism."

Hess' stated that "50% of the breast-fed infants show more or less tendency to rickets in some form." He further states that it is most marked in winter, and that colored infants are much more prone to rickets than white infants.

Scurvy, in connection with vitamin studies, has again become a subject of investigation, and of much importance in its relation to pasteurized milk. The pathology of scurvy has long been known. Line (1772), in his "Treatise on the Scurvy," included a chapter on "dissections" and a postscript on "Appearances on Dissection of Scorbutic Bodies." Barlow (1883) established the identity of scurvy of adults and infants, and contributed much to the study of pathology of the disorder.

Hess describes the bone lesions (which are considered diagnostic) as follows:

"All the bones are rarely affected by scurvy, and those which are affected show lesions to a varying degree. One of the peculiarities is that it involves the junction of the diaphysis and the cartilage. The bones most apt to show typical lesions are the ribs. \* \* \* \* \*

"The osteo-chondral junction is greatly swollen, somewhat beaded and when cut longitudinally shows on gross examination a transverse yellowish bar, corresponding to the disorganization described below.

"Under the microscope, the line of junction is not sharp and straight as is normally the case, but presents a wavy contour, the cartilage jutting into the rib, instead of abutting in neat apposition to it. The bone is hollowed out and irregularly concave; whereas the cartilage presents a convex appearance.

"At the site of junction is the Truemmerfeld area, where the normal tissue is splintered and fragmented. Everything is in a state of disorder—trabeculae of bone of various shapes and sizes lie scattered about, the cells irregularly arranged and much distorted, signs of recent hemorrhage, unrecognizable detritus.

"The picture is that of a weakened bone having been crushed by the pressure of the more compact cartilage. Higher magnification shows that there are few osteoblasts (generally associated with the deeper fragments of bone), a varying number of intact red cells, according to the occurrence of hemorrhage, and occasional spindle- and star-shaped connective tissue cells. Covering

this mass of detritus there is frequently more or less protective fibrin which has undergone some hyalin or connective tissue organization.

"The cartilage is also not normal. Its cells do not present an orderly arrangement, the proliferating columns having disappeared in the central convex portion, and being present to a varying extent near the periosteal border. \* \* \* \* \*

"Below the Truemmerfeld area is the framework marrow, another distinguishing feature of scurvy. This extends for 5-10 mm. towards the lymphoid marrow, where it ends more or less abruptly. It is composed of a loosely constructed fibrillar tissue on a gelatinous appearing ground work, of sparsely scattered cells, and bony trabeculae which are normally thin and weak. Here and there are black pigments and hemorrhages, especially adjacent to the Truemmerfeld. \* \* \* \* \*

"The lack of bony structure and rarefaction clearly is not due to an increase in the number or the function of osteoclasts, for these do not appear in excess. It is merely normal bone resorption with lack of bone regeneration. \* \* \*

"Hemorrhage occurs in bone as elsewhere \* \* \* chiefly beneath the periosteum, rarely beyond the epiphyseal line. As a result of lack of bone formation and the consequent weakening of the corticalis and the spongiosa, frequently a separation of the diaphysis from the epiphysis results. This lesion should not be regarded as a true separation; for, as Barlow has pointed out, the line of cleavage is not at the junction, but below it, involving the uppermost region of the diaphysis. In some instances cartilage is telescoped into the crushed end of the bone."

Hess points out that the chief difference between the bone lesions in rickets and scurvy is that in rickets there is found, at the epiphyseal junction, a broad band of incompletely formed ossified tissue not found in scurvy. In scurvy, growth is inhibited, but the growth that does take place is in an orderly manner. There is also a paucity of blood vessels in the cartilage and marrow in scurvy, and an increase in vascularity in rickets.

Kramer, over 200 years ago, on the treatment of scurvy, writes:

"Seek the cure of scurvy neither in the armamentarium of the physician nor in the apothecary shops. The druggist will be of as little aid to you as the art of the surgeon. On the other hand employ fresh vegetables, the juice of fresh antiscorbutic plants, oranges or lemons, or the juices of those fruits preserved in sugar; in this way, without other means, you will be able to overcome this terrible disease."

It is now accepted that the cause of scurvy is due to the lack of an unidentified factor which has been termed vitamin C, which is present in the citrous fruits, green vegetables, sprouting grains, and milk to a varying degree, depending on the diet of the cow, and the methods involved in the handling of the product, its age and treatment. Potatoes and tomatoes are two of the more important sources of this vitamin. Cabbage subjected to a temperature of 90-100°C. for 20 minutes (Hess<sup>7</sup>) lost 80% of its antiscorbutic vitamin and 1.5% was lost at 10°C. This is of much importance in the use of milk in infants.

Shipley, McCollum and Simmons<sup>8</sup> have reported that rats with uncomplicated beri-beri, induced by diets deficient in

vitamin B, manifested the same lesions as guinea pigs with scurvy:

"The microscopical picture of these bones was quite indistinguishable from that seen in the bones of guinea pigs with scurvy. The bones were very osteoporotic. One received the impression that all growth must have stopped. The epiphyseal cartilage was very shallow (only 4 or 5 cells in depth) and stained intensely with basic dyes. This was true of the cell bodies as well as the matrix. The cells in the marrow proliferative zone were very much flattened in the direction of the long axis of the bone. The intercellular substance of the cartilage was very abundant, and in the shallow zone of provisional calcification it was heavily infiltrated with lime salts. Here and there at intervals thick heavy rods of intercellular substance projected from the medullary border of the cartilage into the marrow cavity which was elsewhere in contact with the calcified provisional zone. \* \* \* \* The blood vessels of the medullary cavity were crowded with red blood cells. In many places the vessels had ruptured and numerous large and small hemorrhages were to be found, especially in the vicinity of the cartilage. In some bones the active bone marrow had been almost entirely replaced by these hemorrhages. Here and there at wide intervals small thick trabeculae or trabecular remnants were to be found scattered through the hemorrhagic area which represents the marrow. These were completely calcified and were surrounded by an endosteum which consisted of a broken layer of osteoblasts, and a fine network filled with mononuclear cells."

#### EXPERIMENTAL

The material used for the pathological studies came from two sources. The larger number of studies were made on animals placed on experiment with the special object of producing and later curing "posterior paralysis." The same symptoms, however, developed in a number of pigs on an experiment initiated to study the toxicity of cottonseed meal. This furnished further material for pathological study.

For the study of "posterior paralysis" a basal ration was used made up as follows: Yellow hominy 200 parts, standard wheat middlings 100 parts, and casein 4.5 parts. This was made into a slop with an equal weight of skim milk,—raw or pasteurized, as will be specified. Five lots of six pigs each were placed on the experiment. Those furnishing material for pathological study were lots I, II and IV. Lot I received the basal ration using raw skim milk, and supplemented with approximately 2.5% of a mineral mixture consisting of equal parts of calcium carbonate and bone meal. Lot II was fed similarly to lot I except that pasteurized skim milk was used. Lot IV was fed similarly to lot II with the omission of the mineral addition.

The pigs in lot I weighed around 40 pounds at the start. They grew fairly well for about a month, with the exception of one pig that remained undersize, and was killed by bleeding from the carotids. The autopsy revealed numerous lung worms (*Strongylus paradoxus*) in the smaller bronchi, solidified lungs,

and chronic nephritis. In five weeks stiffness was observed in one of the pigs, and about the same time a loss of appetite, a roughened coat, and a condition of lassitude were also observed. Later the pig became lame, the hind legs being so weak that the animal could not stand without effort. Several days later this condition was manifested in two other pigs; and on the sixth week of the experiment the remaining two were in the same condition. The disorder manifested itself much in the fashion of an enzootic, in that the pigs did not all become stiff at once. Two pigs in this lot were killed by bleeding from the carotids, and furnished material for study. One died apparently from constipation. Two were restored to health by the addition of alfalfa.

The pigs in lot II remained normal, making fair gains for two months, with the exception of one pig that died of intestinal trouble, and a pig that was removed because of failure to make normal gains. About the third month of the experiment stiffness was observed in the remaining pigs. Hemorrhage in the mouth was apparent in one of the pigs. Severe sweating under the ears, rough coat, and a frenzied squeal were the other symptoms revealed. These pigs were all killed in the advanced stages of the disorder and carefully autopsied. Specimens of bone and affected organs were taken for microscopic study.

In lot IV pig 336 made fair gains for one month after which time it failed to gain. - It was killed by bleeding from the carotids, and upon autopsy revealed numerous intestinal worms (*Ascaris lumbricoides*). The bones, however, showed lesions which resembled those in the others. They will be discussed later. Pig 303 in lot IV went stiff after being on the experiment three months. It was cured with cod-liver oil, and then slaughtered.

The pigs that became available from the cottonseed experiment previously mentioned were fed the following ration:

Yellow hominy	58 parts
Cottonseed meal	25 parts
Middlings	10 parts
Molasses	5 parts
Calcium carbonate	1 part
Bone phosphorus	1 part

This ration was fed twice a day and the consumption was recorded. As the pigs became stiff their appetites became abnormally poor, and the feed was cut down accordingly. It is quite evident that this ration is lacking in vitamin C. The mineral supply is adequate so far as can be ascertained from the

available data. When eating normally the pigs received about a half-ounce of calcium a day. The source of vitamin A is in the yellow hominy, which probably contains the antirachitic vitamin (which Mellenby<sup>9</sup> concluded was vitamin A, but which the more recent work of McCollum<sup>10</sup> would indicate is another factor).

These pigs were all stiff within two months. Four were autopsied and showed lesions similar to those in the other pigs. The bones were similarly affected. The five remaining pigs were given 30 spoonfuls of tomato juice a day, starting February 15, when all were in the very late stages of the disorder. This was changed to alfalfa and potatoes (5% alfalfa being added to the grain mixture and two potatoes apiece cut up and fed with the ration) five days later. After two weeks the potatoes were discontinued. Minerals were fed *ad libitum*, but the pigs appeared to have made marked improvement before these minerals were consumed at all. Within two months these pigs were normal as far as could be seen.

#### PATHOLOGY

The affected organs of eleven pigs have been studied, as well as the thyroids and adrenals. The bones of ten pigs have also been examined. The technique employed in the preparation of bone has been previously described.<sup>13</sup>

In all cases the adrenal and thyroid glands were normal. The livers showed passive hyperemia. The kidneys showed changes which were rather uniform. There was hyperemia in the glomeruli, albumin casts in the convoluted tubules with slight cloudy swelling, dilation of the collecting tubules with lymphocytes and fibroblasts between the tubules in the zona radiata of the cortex. The most marked change in the digestive tract was hyperemia. The spleen in most cases was normal. The heart was normal in all but one case. In this case the ventricles were distended and the myocardium was degenerated. Infection was apparently the cause, for the animal also had an acute, exudative, cerebral meningitis. Where lung worms were present there was an exudative pneumonia. The lymph glands, in all cases, were congested. Routine clinical examination of the blood showed no leucocytosis and no anemia.

The bones, in which the most marked changes were found, were in many cases all involved. The head of the femur was in all cases affected more than other bones. The changes there, on gross examination, were the presence of dark areas in both

the epiphysis and diaphysis, an irregular boundary on either side of the epiphyseal cartilage, and in many cases a separation at the epiphyseal plate. The epiphyseal cartilage was wider

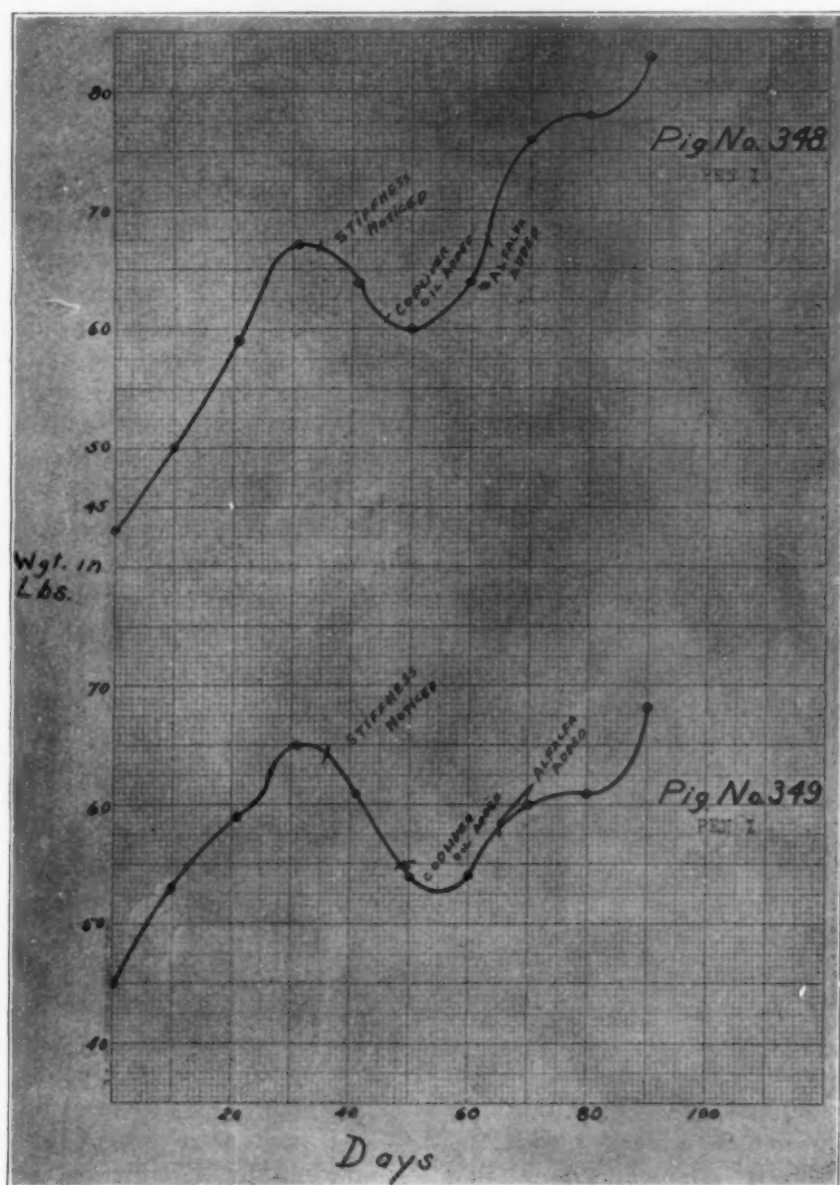


Chart of pigs 348 and 349 showing gains in weight before and after stiffness developed and after the trouble has been removed. Note the marked improvement on addition of alfalfa.

than normal. The bones were softer than those of pigs of the same age, not on the experiment.

The bone marrow appeared red on gross examination. Microscopically it showed an increased number of red cells and an increase in hemopoietic activity. This was constant in all cases examined.

The osteo-chondral junctions of the ribs were enlarged. In four pigs these were studied under the microscope.

The changes in the distal end of the femur were as follows:

*Pig 77 from lot I:* In the diaphysis the bony trabeculae were atrophied. The spaces between these trabeculae were filled with embryonal connective tissue and blood vessels. Hemorrhage was evident in the area immediately under the epiphyseal cartilage, and also in other parts of the diaphysis. The epiphyseal cartilage was very irregular, and in one area hemorrhage and connective tissue had replaced the cartilage. Hemorrhage was present to such an extent under the cartilage that the bone proper was separated from the cartilage, leaving a space between them (fig. 3). In the epiphysis the subchondral bone was replaced in places by embryonal connective tissue and blood vessels, together with hemorrhage in spots. The extent of this granulation tissue was from the articular cartilage to the epiphyseal cartilage, extending two-thirds the distance. The articular cartilage in this specimen did not appear to be involved.

*Pig 342 from lot I:* In the diaphysis the bony trabeculae were atrophied; and the embryonal connective tissue and blood vessels replaced the marrow spaces, with large areas of hemorrhage in evidence. Some cartilage was also present in the diaphysis. Immediately under the epiphyseal cartilage hemorrhage was present to such an extent that a separation of the diaphysis from the cartilage took place. This was not at the cartilage, but immediately below. The border of the epiphyseal cartilage was very irregular. The cartilage was greatly thickened, in places being thirty cells in thickness. This would indicate an interference in the normal process of calcification, and consequently an increase in the amount of cartilage present. In the epiphysis, bordering the epiphyseal cartilage, hemorrhage was present to a marked extent. Marrow elements were present but showed an increased activity, as indicated by a reduction in the amount of fat and an increase in the number of blood forming cells. In places there was absorption of bone, in which areas hemorrhage was present, and was perhaps the cause of the



Fig. 1.

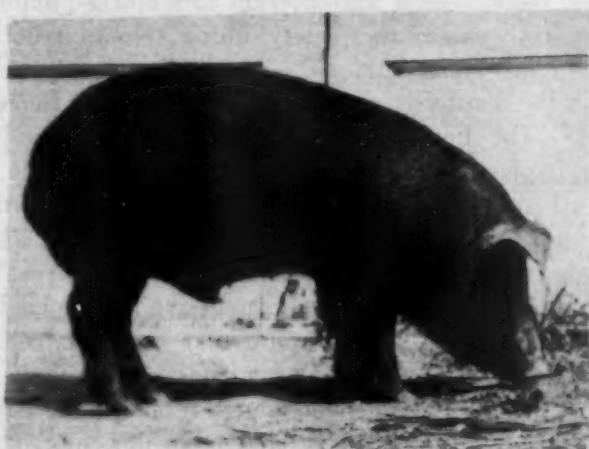


Fig. 2.



Fig. 3.

## PLATE I

FIG. 1—Fig 349 showing "posterior paralysis."

FIG. 2—Fig 349, after being cured with alfalfa.

FIG. 3—Head of femur, pig 377, lot 1. x 13. A, normal bone of epiphysis; B, epiphyseal cartilage; C, space between the epiphyseal cartilage and diaphysis lined by hemorrhage (E); D, bone of diaphysis showing the marrow spaces filled with granulation tissue; E, hemorrhage

absorption. Toward the articular cartilage the subchondral bone was replaced to a large extent by embryonal connective tissue, blood vessels and some hemorrhage. Where hemorrhage was present the articular cartilage showed a changed staining reaction, indicating degeneration. The zone of provisional calcification was irregular, and there was vascularization of the articular cartilage from the subchondral bone. About two-thirds of the epiphysis beginning at the subchondral bone was involved.

*Pig 312 from lot II:* In the diaphysis embryonal connective tissue and blood vessels were present to a marked extent around the bony trabeculae, completely filling the area between the atrophied trabeculae (fig. 8). There was much hemorrhage present in the diaphysis, especially in the region of the epiphyseal cartilage. The border of the epiphyseal cartilage was very irregular, in places vessels from the bone penetrated the cartilage, and in others the cartilage extended below the border. The cartilage cells were flattened in the direction of the long axis of the bone. In the epiphysis the border at the epiphyseal cartilage was very irregular. Vessels and connective tissue penetrated the cartilage in places. Many epiphyseal cartilage cells showed degeneration. Near the articular cartilage embryonal connective tissue, blood vessels, and hemorrhage replaced the subchondral bone in places. Under the cartilage, areas of hemorrhage caused a rarefaction, leaving open spaces in the bone. Degenerated cartilage cells were present in the epiphysis, in the region of the articular cartilage. The zone of provisional calcification was very irregular. There was vascularization of the articular cartilage from the subchondral bone. The periosteum showed hyperemia. The articular cartilage showed areas of irregular staining, marked degeneration and, next to these, areas of proliferation (fig. 8).

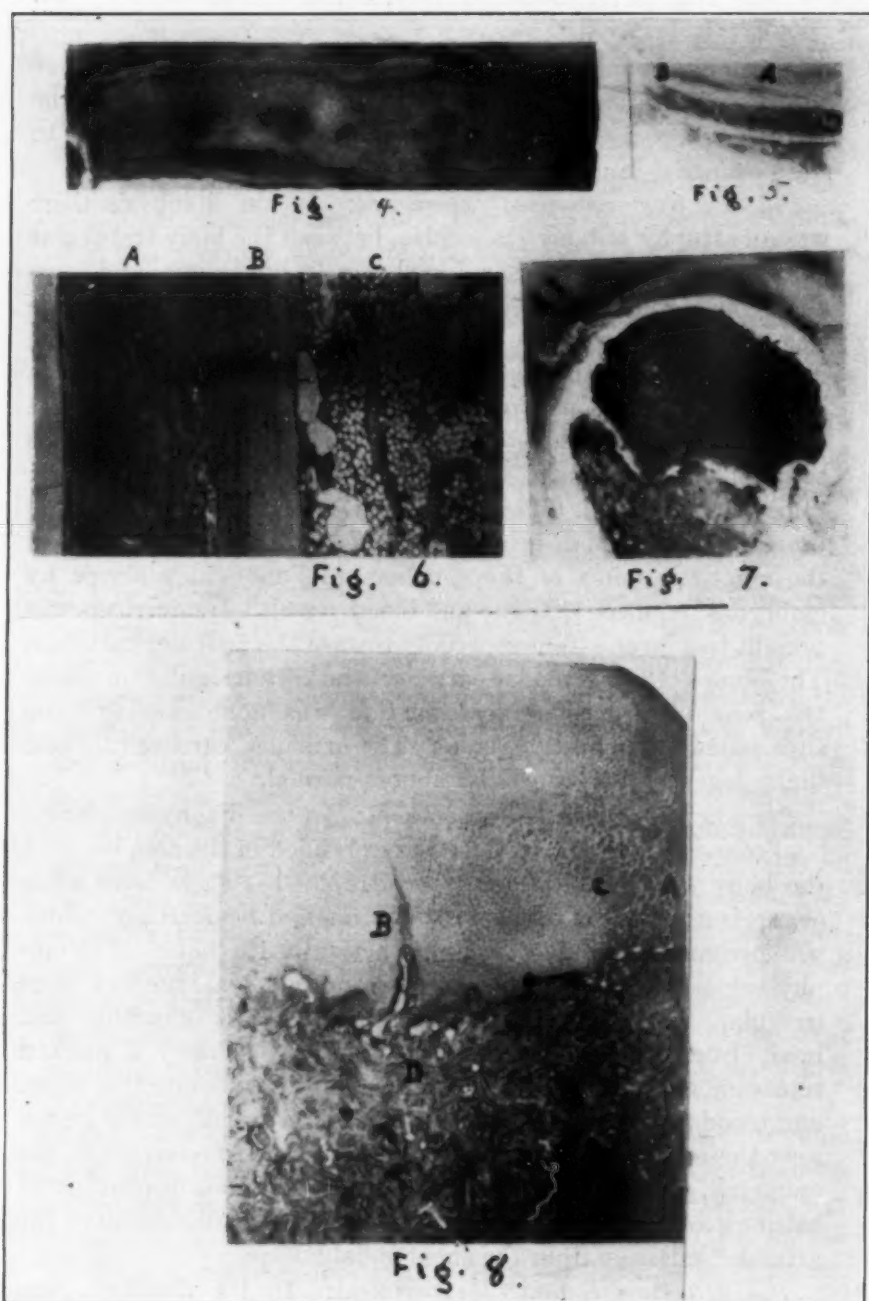
*Pig. 336 from lot IV:* The diaphysis was filled with embryonal connective tissue, blood vessels, and some hemorrhage between atrophied bony trabeculae. Just below the epiphyseal cartilage was an open space, running almost the entire length of the cartilage. On either side of this space hemorrhage was present, which had apparently caused this separation of the epiphyseal cartilage from the diaphysis. The border of the epiphyseal cartilage was very irregular. In the cartilage were present projections of granulation tissue from the underlying bone. In the epiphysis the subchondral bone was replaced in places by embryonal connective tissue, blood vessels and some hemorrhage. The

zone of provisional calcification was somewhat irregular. There was vascularization of the articular cartilage, and in places the cartilage showed degeneration, as indicated by an inability to stain properly, and a disappearance of the nuclei.

*Pig 328 from cottonseed experiment:* In the diaphysis there was an atrophy of bony trabeculae, between the bony trabeculae embryonal connective tissue, blood vessels, hyperemia, and some hemorrhage were present. The border of the epiphyseal cartilage was very irregular, with cartilage penetrating deeply into the diaphysis. Under the epiphyseal cartilage hemorrhage caused a separation of the bone from the cartilage, leaving an open space three-quarters the diameter of the bone. Toward the epiphysis the epiphyseal cartilage was very irregular, and the cells were somewhat flattened. The epiphyseal cartilage was very much broader than normal, in places being 40 cells in thickness. In the epiphysis much of the subchondral bone was replaced by embryonal connective tissue and blood vessels. Hemorrhage was present to a large extent especially toward the articular cartilage. The border of the articular cartilage was very irregular, in places the zone of provisional calcification was gone, having been superseded by connective tissue. The articular cartilage showed slight degeneration, otherwise almost normal.

*Pig 334 from cottonseed experiment:* In the diaphysis, embryonal connective tissue and blood vessels filled in the area between the bony trabeculae, which were atrophied. There were areas of rarefaction in the diaphysis, with marked hemorrhage, which was perhaps the cause of the rarefaction of the bone. The epiphyseal cartilage was thicker than normal, the borders were irregular, and the cartilage contained marrow elements and bone, from the diaphysis. The epiphysis showed a marked replacement of subchondral bone by embryonal connective tissue and blood vessels, with some hemorrhage, especially in the region near the articular cartilage. There was much rarefaction in the epiphysis, with hemorrhage bordering it. The zone of provisional calcification was irregular, and there was a vascularization of the articular cartilage from the subchondral bone.

*Pig 310 from cottonseed experiment:* In the diaphysis was present embryonal connective tissue, blood vessels, and some hemorrhage between atrophied bony trabeculae. There was also much hyperemia present. The border of the epiphyseal cartilage was very irregular, with degenerated cartilage cells penetrating into the epiphysis and diaphysis. The epiphyseal



## PLATE II

FIG. 4—Rib of pig 312, showing what appeared to be echymoses. Natural size.

FIG. 5—Rib of pig 312. Longitudinal section.  $\times 1$ . A, pleural surface; B, subpleural hemorrhage.

FIG. 6—Rib of pig 312. Longitudinal section.  $\times 13$ . A, osteoid tissue formation in the area of hemorrhage; B, normal cortical bone of rib; C, normal medullary bone of rib.

FIG. 7—Head of femur pig 312. Natural size. The dark areas indicate hemorrhage.

FIG. 8—Head of femur pig 312.  $\times 13$ . A, normal articular cartilage; B, degenerated articular cartilage that is being vascularized; C, proliferating area of cartilage cells that is degenerated; D, subchondral bone with the marrow spaces filled with granulation tissue.

cartilage was very much thickened, being 40 cells in breadth in places. In the epiphysis there was a replacement of bone by embryonal connective tissue, blood vessels and some hemorrhage. Hemorrhage was present to such an extent in places that it had resulted in a rarefaction of bone. There was hyperemia in the epiphysis. The zone of provisional calcification showed marked irregularity due to the connective tissue and bone around it. There was vascularization of the articular cartilage from the subchondral bone. The articular cartilage showed areas of degeneration, characterized by an inability to stain properly, and a fading or disappearance of the nuclei.

In the costo-chondral junctions, the changes were somewhat the same as in the femur. They are as follows:

*Pig 377 from lot I:* Under the cartilage, embryonal connective tissue and blood vessels comprised about two-thirds of the area, lying between atrophied bone. Hemorrhage was present in scattered areas. The border of the cartilage was greatly enlarged, as a result of the connective tissue. The bone presented a concave contour, while that of the cartilage was convex.

*Pig 312 from lot II:* Embryonal connective tissue filled in the area between atrophied bony trabeculae, with hemorrhage scattered throughout the bone. Under the cartilage was an area of rarefaction, with hemorrhage present about it. The cartilage showed some degeneration, and apparently an increase in vascularization.

*Pig 328 from lot IV:* Embryonal connective tissue and blood vessels made up about two-thirds of the tissue under the cartilage. At the border between the cartilage and the bone, hemorrhage and connective tissue filled the entire area. The border was very irregular. The cartilage showed some degeneration.

In one case only (pig 312) there was what appeared to be subperiosteal hemorrhage on the pleural surface of the rib (figs 4 and 5). Microscopically these areas showed osteoid tissue formation. This osteoid tissue was grown on to the normal cortical bone (fig. 6).

Two autopsy reports are included to show that occasionally the lesions resemble those found in cases of hog cholera.

*Pig 304 from lot II:* Age three months and two weeks. The morning before death the hog was paralyzed.

*External examination:*

Oral mucous membrane rather pale. Anal mucosa pale.

*Internal examination:*

A. Condition of the animal was rather poor, with little fat remaining.

B. Peritoneum was congested over the small intestines and covered with a little fibrinous exudate.

C. Spleen was normal.

D. Pancreas appeared normal.

E. Left kidney appeared rather pale. Upon section the cortex appeared pale. Medulla was slightly congested. The capsule peeled normally. Numerous petechiae were present on the cortex beneath the capsule. The right kidney showed five petechiae. The capsule peeled normally.

F. Urinary bladder was distended with clear urine. The mucosa was marked by numerous hemorrhages, petechiae and ecchymoses.

G. Liver was fairly normal with slight congestion.

H. Stomach contained a peculiarly smelling, greenish colored material along with a certain amount of mucus. The small intestines contained the same dark greenish material. They also showed some congestion of the mucosae. The cecum and large intestine showed some congestion, and also contained dark solid feces.

I. Most of the lymph glands showed both hemorrhage and congestion.

J. Lungs showed solidification in the ventral part of the cardiac and apical lobes. *Strongylus paradoxus* found in the bronchi.

K. Brain. Marked congestion and a serous exudate in the meninges. The cortical grey matter was pinkish and showed marked congestion.

L. Heart. There was adhesion between the pericardium and epicardium, in spots, by fibrous tufts (partial synechia).

The diagnosis was given as suspicious of hog cholera. The other pigs in this pen showed symptoms similar to this one, but change of diet brought marked relief.

Pig 312. Killed by bleeding from carotids, Feb. 5, 1923.

*Symptoms and external examination:*

Animal stiff, condition poor. Difficulty in rising. Coat rough and sweaty around the ears. Hemorrhage in the mouth. Teeth loose, some decayed.

*Internal examination:*

A. The animal was in poor condition, with very little subcutaneous and subperitoneal fat.

B. The peritoneum was normal.

C. The spleen showed petechiae on surface. On section it appeared normal.

D. The pancreas was apparently normal.

E. The kidneys showed parenchymatous degeneration in the cortex and medulla, also an increase of connective tissue in the hylus.

F. The liver was nearly normal with a slight increase of interlobular connective tissue.

G. The stomach and intestines were normal.

H. The urinary bladder showed scattered petechiae in the mucosa. The wall was somewhat thickened.

I. The lungs appeared normal.

J. The thyroid glands were paler than normal.

K. The heart appeared normal.

L. In the bones, the distal end of the femur showed division at the epiphyseal plate. There was a dark area under the cartilage. There was subperiosteal hemorrhage in the ribs and the ilium and femur showed swelling, perhaps beading.

L. The adrenals were normal.

### DISCUSSION

The principal purpose of the experiment was to identify this disease with one of specific etiology. The pathological changes in the bones are at present the only means of identifying rickets and scurvy,<sup>11</sup> with which this disease is associated. The most marked change in the bones in rickets is the production of osteoid

tissue in abnormal amounts. There is also enlargement and disorganization of the zone of provisional calcification.<sup>4</sup> McCollum<sup>6</sup> also describes a metaphysis composed of blood vessels, osteoid tissue, marrow elements, and cartilage cells in all stages of degeneration. In scurvy the most marked change is at the costo-chondral junction. The cartilage is irregular at the border with cartilage jutting into the bone. Below the cartilage is the Truemmerfeld area in which there is hemorrhage, and occasional spindle- and star-shaped connective tissue cells. The arrangement of the cartilage cells is also abnormal. Below the Truemmerfeld is the framework marrow, another distinguishing feature of scurvy.<sup>7</sup> It is composed of loosely-constructed fibrillar tissue on a gelatinous appearing groundwork of sparsely scattered cells and bony trabeculae. Here and there are black pigments and hemorrhages, especially adjacent to the Truemmerfeld. In the bones of the pigs examined in the above experiment, the most marked change is the presence of granulation tissue and hemorrhage in the diaphysis and epiphysis of the femur, and in the diaphysis of the rib. There is also a thickening of the epiphyseal cartilage, and an imperfect arrangement of the cartilage cells, with cartilage present in the diaphysis of both ribs and femur, to a varying degree. Osteoid tissue, except in one case reported, was not present to any great extent. The picture might be considered to have points in common with that of scurvy, as the cartilage jutting into the bone in the rib, the presence of hemorrhage, and the presence of connective tissue, except that Hess describes his connective tissue as "loosely-constructed fibrillar tissue on a gelatinous appearing groundwork." In the case of the pigs in the above experiment, the connective tissue was of embryonic nature, rich in cells and therefore more or less compact. The region under the cartilage of the ribs might be considered a Truemmerfeld area, for in it are hemorrhage and spindle-shaped connective tissue cells. In support of the hypothesis that the disease is rickets, is the fact that the epiphyseal cartilage is thickened, and also that there is a lack of calcification, as evidenced by the atrophy of the bony trabeculae. The changes, however, coincide rather closely with those reported by Zilva et al.,<sup>2</sup> in which he reports an increase in proliferating and hypertrophic cartilage cells in the rib; a deep penetration of blood vessels from the bone marrow into the cartilage; and the presence of fibro-cellular tissue instead of

marrow. In our pigs there was hemorrhage in addition to these changes.

To draw conclusions as to the identity of this disorder with a specific deficiency disease is not possible. The pathology of rickets is worked out on rats, while that for scurvy, on human beings and on guinea pigs.

Findlay and Mackenzie have reported the following condition of bone marrow in guinea pigs with acute scurvy: "congestion with hemorrhagic foci, though degenerative changes in the hemopoietic tissues are not noticeable." This condition is comparable with that observed in the pigs.

The pathological condition observed in the pigs was greatly influenced by factors other than the diet. For instance eight out of eleven pigs autopsied in the first experiment showed lung worms. In these cases there was an exudative pneumonia. Many of the pathological changes occurring in these pigs, as revealed by the postmortem examination, are a result of this condition. That the diet played an important part in lowering the resistance against invasion and multiplication of organisms is evident from the fact that in normal pigs slaughtered at the college abattoir, lung worms are often present, but not pneumonia, at least not to the extent found in the experiment pigs.

The parenchymatous organ most uniformly affected was the kidney. That is perhaps explained by the fact that when the pigs became stiff they retained their urine for a long time. It was observed that whenever the pigs were forced to rise they would urinate for a considerable length of time. This explanation is substantiated by the distention of the collecting tubules observed microscopically in almost every case.

The passive hyperemia in many cases was most likely due to pneumonia.

The central nervous system of twelve pigs, examined by the Nissl method, appeared normal. It is evident, therefore, that the symptoms manifested are due to the condition of the bones, the kidneys, or possibly to changes in the peripheral nerve endings. These have not been studied. There was, however, hypesthesia in the skin in the later stages of the disorder.

Since the feeds employed in these experiments are those that may well be considered adequate, the practicing veterinarian may easily overlook it as the primary etiology, and on account of the similarity of the symptoms and the lesions with various stages of hog cholera, there may be an explanation for so-called "breaks"

following the use of anti-hog cholera serum and virus. This also offers one more condition to be differentiated from hog cholera.

Microscopic sections of the femurs and ribs, at the costochondral junctions, from the recovered pigs 307, 348 and 349, taken at the places where the lesions in the other pigs were most constant, showed the zones of provisional calcification straight and uniform, the bony trabeculae normal and where there was apparently hemorrhage previously were areas filled in with fat. In place of granulation tissue between the bony trabeculae, there was now normal fatty marrow. There was also a normal amount of hemopoietic marrow. In only one small area were there young vessels and fibroblasts in the diaphysis next to the epiphyseal cartilage. In this area, however, the amount of connective tissue was much less than the untreated pigs and the bony trabeculae were normal.

#### SUMMARY

1. The essential lesions are an osteoid rather than osseous tissue formation at the epiphyses and costochondral junctions, indicating a lessened amount of calcification somewhat suggestive of rickets, without the usual deformities found in this condition.

2. This is not due to deficiency of calcium and phosphorus in the diet since these were supplied. It seems rather to be due to lessened assimilation of these minerals.

3. Addition of alfalfa to the ration, and possibly also cod-liver oil alone, produces a recovery of cases already affected.

4. The symptoms in the early stages and the lesions in the parenchymatous organs are similar to those that are supposed to be diagnostic of hog cholera.

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#### DISCUSSION

DR. KERNKAMP: I believe there is a disease of swine characterized by paralysis that is a deficiency disease, and I think it is a mineral deficiency. I have seen cases that come down apparently so suddenly that it is hard to believe that it was a deficiency disease, for the reason that so many in the herd developed it at about the same time.

This disease attracted our attention in Minnesota a few years ago. We received numerous inquiries, and decided to investigate it. After making a few field trips and taking vitamins into consideration, they seemed to be a little out of the question. Some foodstuffs apparently contained a sufficient amount of the vitamins, or they were foods that should have contained the vitamins. In other cases, it would be easy to say, "Well, here is a vitamin deficiency. The foods they are receiving are foods which they tell us are deficient in certain vitamins." But when summing up the whole thing, we began to look at it as a mineral-deficiency proposition, a winter-feeding proposition, as Dr. Goldberg brought out.

The first thing we wanted to try was calcium, and we fed foods that, as far as we could determine, would be deficient in calcium carbonate. We kept pigs solely on yellow corn and water, and checked this with others fed calcium carbonate (2%) in the ration.

The pigs that received only the corn developed a stiffness, some of them a paralysis, exactly as shown in the picture which Dr. Goldberg passed around. In others, the front legs were involved, that is, they would knuckle over at the knee and pastern.

Of the pigs that received calcium, one of them, in a lot of seven, showed this disease at postmortem. We have carried the calcium idea through, mainly with the thought of trying to determine what causes the disease.

We have started this last year with phosphorus. Most of the foodstuffs that swine receive contain more phosphorus than calcium. Cereals, particularly, are deficient in calcium, but usually contain a considerable amount of phosphorus.

In connection with this, we made a field inspection trip that I thought was very interesting. On one farm, there were pure-bred Chester White pigs. This man had in the neighborhood of 60% of his pigs involved with stiffness, knuckling over, and symptoms of paralysis. It was noteworthy—as we went through these pastures—to see how they had rooted the pastures, "in dirt to their eyes," so to speak, rooting and getting into the dirt as much as they possibly could.

The veterinarian with me said, "We'll go over to another pure-bred breeder here and see his conditions." The farm was about a quarter-mile away. On this man's place, there were about an equal number of hogs, but none of the pigs was sick. They were in an alfalfa pasture. We looked about the place carefully, and found that in certain places in the feeding lots, he kept bone-meal. We said, "Do your pigs eat much of this?"

He said, "They devour a lot of that bone-meal."

It occurred to us right there that we were dealing with a deficiency, and probably a mineral deficiency disease.

I feel like Dr. Goldberg, in that I don't know whether it is rickets, whether it is scurvy, osteoporosis, or osteomalacia, but that it must be at least some osseous disease. Our X-ray plates show that in many cases there is a marked deficiency of calcium in the bone, and I was interested to hear what Dr. Goldberg said regarding the lumbar region. On our X-ray plates, we found the lumbar vertebrae (because in these paralyzed pigs, one looks immediately to the lumbar region) were not deficient in calcium as compared to the bones of the appendicular skeleton.

DR. PICKENS: I would like to ask Dr. Goldberg one question: What were the lesions of the cord over the lumbar region and farther up, if that was examined?

DR. GOLDBERG: There were no lesions of the cord. We sectioned the cord in these diseased animals, animals that have recovered, and animals that were not in the experiment, but in the slaughter-house, and we have found them all alike. If I found a little thing out of the way, a little blood vessel or something in there, I would go to the neurologist and find out whether that was normal or not, and he assured me it was perfectly normal.

CHAIRMAN GOSS: In this connection, I would like to refer to some experiments that were conducted in Kansas, on feeding pigs corn and water, as Dr. Kernkamp stated. The pigs there did not go down. However, sows that were heavy with pigs did go down, and they were unable to get up. The

water in Kansas contains a large amount of mineral—lime—and it is exceedingly hard. Goiter is of rare occurrence in that district. That would point possibly to a better supply of certain lime salts.

DR. ATHERTON: In an investigation of such cases and in offering treatment, I find that it is practically necessary to encourage the use of lime salts in the diet, because you will nearly always find it, where lime salts are denied, and it is necessary also to add alfalfa, clover, cow-pea hay, or something like that, to the diet to obtain the results after you have added the lime salts.

DR. GOLDBERG: The first question that was brought up was in regard to the deficiency of calcium and phosphorus, and vitamins, perhaps. In all these rations, we supplied a sufficient amount of both calcium and phosphorus to these animals. If there is a deficiency, it is rather something that prevented the metabolism of the calcium and phosphorus than a deficiency of the calcium or phosphorus.

Regarding the question of the vitamins—in food, we know a good deal about the carbohydrates—I mean the actual chemical composition. We can produce some of them. We know a good deal about the fats. We can take them apart. We can find out exactly what status the fat is in, how it will combine with iodine and various other elements. But the protein molecule, although it has been studied for a long time, we don't know very much about. The first attempt at trying to separate the protein molecule, in order to determine the actual chemical composition of it, was made in 1810, by Wollaston, and he succeeded by hydrolyzing, that is, by adding water to the molecule together with other chemicals that would make the molecule take up water, in obtaining an amino-acid cystine. It is a very complicated amino-acid. The least complicated amino-acid is glycocoll, and it is not necessary to supply glycocoll in the body, because the body can synthesize glycocoll from other amino-acids.

Then we have a series of amino-acids that have been discovered by one or another. In connection with these other amino-acids that have been discovered, I want to mention Paul Ehrlich, who discovered one, and Emil Fischer. Emil Fischer did a considerable amount of work on the proteins and on the amino-acids, and he also succeeded in putting the simpler amino-acids together to form a simple protein. He had a combination of, I think, fourteen, but they were made up of just two amino-acids, glycocoll and leucine. The last one that was found—I think it was in 1922—is a combination of two amino-acids, and just where they combine is not exactly known. The name of this is glutathione, that is, it is a combination of glutamine and cysteine.

As a result of this latest discovery, it is quite possible that our conception of metabolism will change entirely. Our idea of how the food materials break up to form carbon dioxide and water is not all as simple as we originally thought.

Then, of course, you all know about the vitamins that have been talked about so much, but about which very little is known at the present time. This particular condition may have to do with some of those amino-acids that may have to do with calcium assimilation. (Applause.)

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### NEW BULLETIN ON ABORTION

"Infectious Abortion of Cattle" is the title of Bulletin 304, of the Ontario Department of Agriculture, by Dr. C. D. McGilvray, Principal of the Ontario Veterinary College. It contains a great deal of information on infectious abortion that every owner of cattle should have. Veterinarians, too, will undoubtedly find much of interest in the publication.

## SOURCES OF INFECTION IN PRIMARY OUTBREAKS OF HOG CHOLERA<sup>1</sup>

*By I. K. ATHERTON, College Park, Md.*

In the investigation of outbreaks of contagious and infectious diseases of animals, not only the most interesting, but undoubtedly the most important part of the history is often overlooked. I refer to the source of infection, or the manner in which the causative agent of the disease was conveyed to or obtained by the affected animals.

As a rule, in such investigations, the only history of the case that any effort is made to obtain is that which would aid in making the diagnosis. After the diagnosis has been made and instructions given for the treatment and care of the animals, the investigator goes on his way, contented with the idea that he has fulfilled his duty in the case and satisfied that he has taken the necessary action to combat the disease, but without a thought for its prevention in the future.

We recently noted an article, by an eminent veterinarian, from which we culled the following paragraph which, we believe, emphasizes our contention.

"Pathogenic organisms are the primary causes of the infectious diseases, and, therefore, the struggle against these diseases resolves itself into a campaign against the causative micro-organisms. In order to carry out successfully such a campaign, it is essential that we know the location of the enemy, the ways by which we may reach it, and we must also be familiar with its strong and weak points. It is a well-known fact that it is better and simpler to prevent diseases than to cure them, and our principles of disease control should be so directed."

The good doctor did not offer anything new in this regard, but he surely reminded us of some things that we have been overlooking, to a great extent. He made it plain that we cannot wage war on any contagious disease in an intelligent manner without certain specific data regarding the causative organism. This does not necessarily imply that the causative agent must be isolated and identified. The point is well illustrated by our many successful campaigns against foot-and-mouth disease. However, we should be able to determine the way in which

<sup>1</sup>Presented at the sixtieth annual meeting of the American Veterinary Medical Association, Montreal, Canada, August 27-31, 1923.

the infection is introduced, how harbored and the manner in which it is disseminated from the original or primary outbreak.

This is the 90th Anniversary of the appearance of hog cholera in the United States. It is passing strange that a disease which is not only preventable, but in reality largely self-eradicating, could cause such enormous losses, year after year and, after an existence of nearly a century, still be one of the most destructive diseases of our domestic animals. I do not know of another disease that has been considered with such awe and has been surrounded with so much mystery as hog cholera. Undoubtedly this deplorable condition is largely, if not entirely, due to lack of information regarding the source of infection in new outbreaks.

It appears strange that some of our leading investigators have been quoted as discouraging efforts to eradicate hog cholera for the reason that the causative agent had not been isolated and identified. Their contentions seem to indicate that there is so much about the disease which is not known that we are not justified in attempting eradication. If we had considered foot-and-mouth disease in this light, no doubt we would be in a sorry plight at this time.

Exhaustive experiments have been carried out to determine the ways in which hog cholera is spread from the primary outbreak, but few investigators have endeavored to find out the manner in which new outbreaks are started.

#### RELIABLE DATA DIFFICULT TO SECURE

Gathering such data often taxes the ingenuity of the investigator to the utmost; in fact, every field man does not prove a success in this phase of the work. The man qualified to obtain such data is one who has the ability to convert himself into a "human interrogation point." In attempting to collect such information, it is often found that the owner or caretaker of the animals deliberately tries to mislead the enquirer. In other cases the outbreak is of such long standing that incidents connected with the source of infection have been forgotten, and it is necessary to refresh the memory of the owner by suggestions as to what might have happened to bring the infection to his place. Then, again, on other premises the animals receive such poor care, and so little is known about them that it is impossible to get any information of value.

It is generally agreed that the introduction of sick or exposed

hogs into uninfected areas is responsible for a certain percentage of the new centers of infection. In some states, this is possibly the main source of infection in new outbreaks. Our experience leads us to believe that the purchase of swine from commission houses, peddlers, and the buying of stocker hogs, especially those obtained at public stock yards, are the greatest sources of danger.

It is generally admitted that "breaks" following the use of, or, as is usually the case, the abuse of the double treatment are responsible for many new outbreaks. The veterinary profession seems to be divided to a certain extent on the subject of breaks following the administration of the double treatment; but even the most radical double treatment enthusiast admits that a certain percentage of these breaks is hog cholera. One of our foremost authorities on swine diseases makes the statement that "To say the least, the use of the simultaneous treatment is contrary to good sanitation." He surely had in mind the breaks following the administration of the double treatment when he penned that sentence.

It appears to be the concensus of opinion, but without any data to substantiate the claim, that there must be many other sources of infection about which we know little or nothing. Information that would clear up this point would mean a big step in the eradication of the disease.

#### GARBAGE LONG A SOURCE OF TROUBLE

In a report, dated February 28, 1880, Dr. Detmers,<sup>1</sup> in referring to the feeding of hogs affected with cholera, stated that animal food in some cases appeared to have a beneficial effect, but added, "Still, feeding animal food constitutes, by no means, a sure protection, because hogs fed in slaughter-houses, and hogs fed with offal from a hotel table, (for instance) those belonging to the Doane House, in Champaign, Ill., in the fall of 1878 became affected and died of swine plague." This is the first reference we have been able to find where garbage feeding is associated with disease in swine.

The first published information which I have been able to find relative to extensive losses among swine fed on garbage appears in a report by Dr. V. A. Moore,<sup>2</sup> in 1907. He describes a condition among swine in the state of New York, in which thousands of hogs were lost and the trouble was diagnosed hog cholera. However, he insisted that the losses were not caused

by a specific disease, but he was of the opinion that the losses were caused by poisoning due to powdered soap and drugs in the garbage.

In 1921, Dr. C. D. McGilvray,<sup>3</sup> in an article read before this association, giving an account of an outbreak of hog cholera in Canada, in 1911, made the following statement:

"A rather curious and striking feature was that on all premises upon which the disease first manifested itself, the hogs thereon were being fed on uncooked swill, kitchen refuse and garbage obtained from hotels and restaurants. On other premises, in the same districts, where the hogs were not being fed upon kitchen refuse and garbage, the hogs were found to be healthy and remained so unless, and until, becoming infected by either direct or indirect contact, or intermediary means, from premises where the disease had already manifested itself. In the latter cases, information was obtainable that infection had been introduced by such means."

#### HOG CHOLERA IN CANADA

Dr. J. G. Rutherford,<sup>4</sup> in a report for the year ending March 31, 1911, makes the following statement, in reference to hog cholera in Canada:

"The frequency with which the disease makes its appearance among garbage-fed swine, not only in this country, but in the United States, as well as in Europe, has led to the adoption of the belief among veterinary sanitarians that these animals become infected through the ingestion of raw, pork products imported from countries in which the disease prevails."

Outside of Canadian reports, but little can be found in reference to garbage feeding causing disease among swine until 1916, when Dr. R. R. Birch,<sup>5</sup> of the Cornell Experiment Station, presented a paper at the meeting of this association, in which he related experiments which he had made, proving conclusively that hog cholera infection was transmitted by infected pork.

Beginning July 1, 1918, the collecting of data relative to the source of infection of new outbreaks of hog cholera, as well as information regarding the manner in which the infection was spread from these new centers of infection, was begun in Maryland. In starting this phase of the work we had no theories which we desired to prove, but we were seeking knowledge that would assist us, if possible, in conducting the hog cholera work in a more intelligent manner.

The attempt to collect such data was apparently setting sail on an almost uncharted sea. In fact the meager information available at that time led us to believe that almost anything could be expected. Authorities on hog cholera were of the opinion that undoubtedly there were many factors about which we knew nothing, and which were responsible for starting new outbreaks. As we once heard it stated, "New outbreaks sometimes occur as unexpectedly and with as little warning as a clap of thunder out of a clear sky." Because of a belief in the mysterious methods by which the virus might be disseminated we undoubtedly, at times, overlooked the simple solution of many cases. Most of the members of the force lacked experience in collecting such information. In fact we were all beset with fear that we would commit some error that would prove to be at best ludicrous. Some of our experiments in attempting to determine the source of infection in outbreaks were not only intensely interesting but very amusing.

#### HOG CHOLERA FOLLIES

In one instance a farmer was driving past a place where hog cholera existed. The owner of the farm was preparing to kill a pig which had been injured. The farmer was so overcome with sympathy for the pig that he removed it to his own premises. You can imagine what happened.

In another instance an outbreak of hog cholera was investigated on a farm belonging to a colored man. He, of course, was questioned relative to buying pork for his table. His reply was: "Indeed, I did not buy any po'k. No, sah, I did not. I buys some salt meat, some hams and some bacon, but I did not buys any po'k."

In still another instance, a farmer admitted that pork for table use was purchased frequently, but insisted that none of the bones, rinds or scraps were fed to the hogs. Indeed not, they were fed to the dogs. The hogs on the premises, which were properly confined, did not contract the disease. However, five little pigs were running at large, could and undoubtedly did partake of the lunch provided for the dogs. Is a Sherlock Holmes needed in this case?

It is needless to state that we made comparatively little progress during the first six months of our endeavors. The entire force was engaged in an educational campaign, and it was impossible to investigate every outbreak reported. For

this reason we were not always able to differentiate between primary and secondary outbreaks. This, coupled with our inexperience, proved a great handicap in the work. The most that we gained was more or less experience in collecting such data, and, to our surprise, we learned as we became more experienced that the feeding of material containing infected pork is, in Maryland, the most prolific source of infection. Forty per cent of the new outbreaks, where we were able to get a history regarding the cases, were attributed to this cause, and 33% to the introduction of new stock. The double treatment was believed to be responsible for 5% of the remainder. In view of later developments it is plain that our lack of knowledge in the beginning regarding the sources of infection led us to ascribe many outbreaks to conditions that could not have been factors in introducing the virus. For this reason our data on the remaining 22% of the cases are of doubtful value.

#### INFECTED PORK A BIG FACTOR

In 1919 we were able to secure data on the source of infection in about 50% of the primary outbreaks. During this year it was apparent that approximately 62% of these cases were due to infected pork, 20% to the introduction of new stock, 8% to infection being harbored on premises, and 3% to the double treatment. Still our lack of knowledge led us to inject the mysterious in some of the cases, and consequently our data on the remaining 7% was questionable.

In 1920 it was found that apparently 80% of the primary outbreaks were due to infected pork, 12% to the introduction of new stock, 4% to infected premises, 3% to the double treatment, and in 1% the history was doubtful.

In 1921 it seemed apparent that approximately 80% of the primary outbreaks were due to infected pork, 18% to new stock, 1% to double treatment, and 1% to infected premises.

For the first six months of 1923 it was possible to get data on the apparent sources of infection in 90% of the primary outbreaks. It is believed from the histories of the outbreaks that 82% were due to infected pork, 16% to new stock and 2% to the infected premises.

A certain percentage of the new outbreaks of the disease each year was ascribed to infected premises, or, in other words, the infection was apparently harbored on the premises from previous outbreaks. I have included these cases in this paper as they

were noted in our annual reports, and I feared that if I did not include them I would be accused of being inconsistent. However, such outbreaks would really be recurrences of the disease instead of new outbreaks. The infection in these instances was already on the premises, and therefore the manner in which the infection was introduced had been determined. Eliminating the infected premises as a factor, and it is patent that it is not, then we have only three factors which have apparently been responsible for the new outbreaks of hog cholera in Maryland. They are: infected pork fed in garbage, table refuse, kitchen swill and butcher offal, the introduction of sick or exposed swine, and "breaks" following the use of the double treatment.

It is interesting to note that as the seven men working in four different districts acquired experience and became better trained in the work, the number of primary outbreaks ascribed to infectious pork gradually increased from 40% to 84%, striking an average of 82% for a period of four years.

Not long ago I heard a veterinarian discussing this subject make the remark that it was possible that in time we might find other factors that were responsible for the introduction of the infection into free territory. Assuming this to be true, even in view of the probability of the further development of our knowledge in regard to this disease, are we warranted in remaining inactive until this knowledge becomes available? I believe we have already delayed attempts at eradication too long, to the detriment of the swine industry.

#### CONCLUSIONS

In conclusion, when investigating a case of sickness among swine in free territory, where a clear history can be obtained, if the investigator can establish the facts that no infected pork has been fed, that no diseased or exposed stock has been introduced in the herd, and the double treatment has not been used, I believe that a postmortem examination will confirm a negative diagnosis of hog cholera. Further, I believe the control of these three factors will eradicate hog cholera.

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## DISCUSSION

DR. E. M. PICKENS: Mr. Chairman, this is a very interesting paper, and I am awfully sorry that we haven't more people here to hear it. It seems to me that, in a way, it is a new thought along hog cholera lines. I think the average individual has felt that there are a great many sources of infection in the new outbreaks, but in Maryland the results that Dr. Atherton has been getting seem to indicate that the three sources cover a very great percentage of the cases. I would like to hear from Dr. Stange, if he has had any experience along that line.

DR. STANGE: Mr. Chairman, I guess everybody knows that we have had experience with hog cholera in Iowa, but as regards the source of the infection, I feel that we are a little uncertain about that. I assume that the conditions in Maryland are considerably different than ours. We have comparatively large farms, practically no garbage feeding except in a few of the large cities, the hogs being fed almost exclusively on corn and grains raised on the farm. We get outbreaks, however, of hog cholera in those communities, and we can't explain how it got there.

I haven't entirely given up the idea that there may be some insect that occasionally may carry hog cholera. I think the Bureau of Animal Industry, in their experiments, showed without any question that hog cholera can be transmitted in that way. But the question still remains: Under practical conditions, is it transmitted that way to any considerable extent?

I would like to say just another word in connection with the use of the word "eradication." I am one of those who has been objecting to the use of the word "eradication," especially in talking with laymen, and farmers at their meetings, in connection with hog cholera. The reason I don't like to use it is because I believe it doesn't lead to the proper frame of mind. Stockmen, laymen generally, expect results. If you go out and tell them that you are going to eradicate cholera and don't show results in a reasonable time, it is going to injure your support. I don't think we ought to lead the people to think that we are going to do more than we can reasonably accomplish in a rather short period of time. I would prefer to be able to do more than they anticipate rather than less.

We must remember in a state like Iowa—and the same condition prevails throughout a large portion of the Central West—you have occasionally very wide-spread infection. In 1913, we lost practically 25% of our hogs. That means that probably over one-fourth of the farms in the State were infected during that one year. Now, when you go out and talk "eradication" of hog cholera, you are taking on a big contract.

I don't believe we can quite put it on the same basis as foot-and-mouth disease, because that disease had no foothold in the United States, but Canada can. We knew after the investigation of foot-and-mouth outbreaks just where every point of infection was, we could practically put our finger on the virus. But here you have something that has become firmly established, and it is more like going to Europe and saying that you are going to eradicate foot-and-mouth disease, where the disease has been prevalent for years.

Our people have become so accustomed to hog cholera that when you try to get the farmer to do very much, outside of vaccination, he says, "I am too busy; I am going to vaccinate"—and then he goes on about his business. There is a practical side to the question with which we have to contend.

As I often tell my students, the big problem in disease control is not the disease itself but the public. Where the difficulty comes is in handling the public. If they would do just what we wanted, it would be in most cases quite an easy thing to control the disease. It is the public we must get under control first, and then we can control the disease. Hog cholera is one of those diseases which is going to require men who can handle the public in order to get results.

If we do more than control hog cholera in Iowa, everybody will be pleased; but if we don't eradicate it they are not disappointed, and I don't anticipate that we will for a good many years to come.

I think Dr. Atherton's paper is a very excellent paper, and I think it shows

a great deal of study. Studies like this are going to help us to get better methods for controlling the disease. (Applause.)

DR. C. A. CARY: I am right well pleased with the paper. I think it is work that should be done in every state, and possibly a great deal more experimental work done along this line.

I want to say a few words about eradication, as I have had considerable experience in eradicating ticks and tick fever in the South. I just want to say a word about that along the line that Dr. Stange mentions, because I agree with him very definitely in all that he has said.

In the case of tick fever, we have the carrier of the disease, the history of it, and we have a very good knowledge of the etiological factors. That we can eradicate the ticks is very definitely established; if we had complete control of every factor in the eradication, we could eradicate all the ticks and the disease in the South in one year.

Now, what have we done? This is the nineteenth year we have been actively working with tick eradication in the South, and it isn't gone yet. I don't know of a single state that started tick eradication, unless it has been Kentucky, in the beginning, that now has absolutely no ticks.

Now I go back to the statement that Dr. Stange made—Why this prolonged work in the South in getting rid of ticks? The whole thing rests with handling the people.

I want to give you an example. In Winston County, in Alabama, the most mountainous county, where there were probably more moonshiners and probably more law-breakers than any county in the State of Alabama at that time, we went into that county—I can't give you the exact year—to eradicate ticks. I went in with fear and trembling, anticipating the worst opposition from the people of any county that I ever started work in. But to our surprise, we cleaned that county in one year, and we didn't have a solitary arrest or a man fined, and we did it with less money than any county we ever worked. Why? Because for some unknown psychological reason the people just fell to it and did what they should. The officers of the county took charge of it.

Now, just another illustration. In the largest and most populous county of the State, the county in which Birmingham is located, Jefferson County, we have been working six or seven years and probably will get through this year. It has cost the county something like \$300,000, and we will have to keep a number of men in there to keep it free when we get it free.

The differences in the human factors are the things. You can readily see that iron miners, coal miners and an immense number of steel factory men are a class of men hard to control in Jefferson County.

Any man who has worked on the eradication of a disease as simple to eradicate as tick fever and tick, will readily see the trouble you are going to have when we get at hog cholera eradication.

DR. ATHERTON: It appears that I don't quite have the faculty of making myself understood in my papers. In other words, they don't quite seem to catch me.

Dr. Stange refers to the different conditions in Iowa and Maryland. I am familiar with both. To a great extent, there is a difference, but not all one way. The people appear to think that we don't have hogs in Maryland to any great extent. Maryland stands eighth in the number of hogs per square mile, which I think is the only way we can figure density of population. We undoubtedly have more garbage-feeding plants than Iowa. However, from my experience in Iowa, I believe the farmers of that State depend more on packing-house products for the meat for their table use than the farmers of eastern states.

Dr. Stange refers to the possibility of an insect carrying the infection. The only way I can answer that is to say that in the six years we have been working in Maryland, we have never yet had the disease spread from a primary outbreak where it was promptly reported. If insects carry the infection, why wouldn't they function regardless of whether the authorities or the neighbors knew of this center of infection?

Dr. Stange said he did not like to use the word "eradication" in connection with work on diseases. I am not talking to farmers at this time. However,

I don't know of any other disease that cleans itself up as nicely as hog cholera does. If Dr. Stange or anybody from any other state will stop to think, I believe he will agree with me that, outside of garbage-feeding plants, where infection is maintained year in and year out by the use of the double treatment, there isn't any part of the state but what is free of hog cholera at one time in the year. It is not eradication of the disease that puzzles me, but it is prevention of the re-infection of free territory.

Dr. Stange said that there was not much connection between foot-and-mouth disease and hog cholera work, or something to that effect, and that foot-and-mouth disease was not widespread. The last outbreak spread to twenty states in thirty days, which we must insist was a widespread dissemination of the infection. Foot-and-mouth disease doesn't usually eradicate itself from any territory. The infection is more easily disseminated than hog cholera. To my mind, a close study of the two diseases shows that the work would be far easier in hog cholera than it would be in foot-and-mouth disease.

Dr. Stange also referred to the matter of getting favorable public opinion. I don't know of farmers in any state whose good opinion it is more easy to obtain than in Iowa. If any man doing field work goes before the farmers and lays his cards on the table, he can get public opinion with him every time.

I don't want anybody to understand that we are going to eradicate hog cholera overnight, by legislative methods, as we eradicated booze from the United States. Dr. Stange has admitted that they don't know much about the source of infection in Iowa. I deem it exceedingly regrettable that in but few states do they have any data regarding the source of infection, and I believe the sooner we start acquiring that data, the better it is going to be for all concerned, because if we cannot tell a farmer how to prevent the infection getting to his place, we certainly are not in a position to give him much information.

There isn't anything that we recommend to the farmers of Maryland to do, to prevent hog cholera from getting to their place, that is going to cost them a penny to try. We have carried on the most extensive educational campaign this year that we have ever conducted, and we have had a fifty per cent drop in the number of outbreaks. The results in any one year may not amount to much, but during the time the Maryland plan has been in operation we have had, with the exception of a slight rise last year, a gradual reduction in the number of outbreaks of cholera in the State. But I do hope that, as sanitarians and officials charged with the protection of the swine industry, we can see where we are making a mistake in continuing to permit ourselves to be held accountable for these appalling losses. Because, if the farmers are following our advice, that is, depending solely on immunization for the protection of their swine, and the disease continues to show a gradual increase, then we are to blame. But if we start a campaign to acquire data regarding the source of infection in the disease, then we will be able to tell the farmers how they can prevent the infection from getting to their place. We can also add that we have no objection to their using any additional methods of protection that they might want to use, so long as they are employed in a manner consistent with sanitation. If we do that, we not only will shift the responsibility, but we can get public opinion with us. Thank you. (Applause.)

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### OHIO ANXIOUS TO TEST

The area plan of tuberculin testing is progressing rapidly in the State of Ohio. It is reported that petitions are coming in for the testing of townships and counties in such numbers that the State and Federal forces cannot take care of them. Twelve counties are being tested and twenty-nine additional counties have made applications to have the work started.

## CAN HOG CHOLERA BE ERADICATED?<sup>1</sup>

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In answering the query contained in the title of this article, it might be stated that in the thought of eradicating hog cholera there must be taken into consideration (1) the need for such action, (2) the means to obtain the end, and (3) the practicability of applying these means.

Speaking of the first factor to be considered, there is no one dissenting in the opinion that the total elimination of hog cholera is greatly to be desired. The United States has produced, since 1912, an average of 66,000,000 hogs a year, with an estimated value of \$860,000,000 annually. An industry that brings a revenue of almost a billion dollars a year should receive all possible protection against losses and depreciation. Hog cholera is still the greatest enemy of the industry and kills many more hogs than it should. In fact, it should not be allowed to destroy a single animal. While many hogs die from various ailments other than hog cholera, and from exposure or lack of attention in housing and feeding, hog cholera is still the most detrimental factor to the profitable raising of swine. Then, we are agreed that the eradication of hog cholera is much to be desired.

What are the practical means to assure the absolute elimination of hog cholera infection? Provided the manner in which the virus is carried from place to place is materially the same as has been preached for a number of years and the infection may live in secluded spots for a long period of time, then hog cholera may be classed as being of a more serious nature than foot-and-mouth disease, since it is a much more fatal ailment. Consequently, measures no less drastic than have been employed to eradicate foot-and-mouth disease would be necessary to eliminate hog cholera. If such measures were followed, it would require the strict quarantine of all infected territory, which would mean virtually the quarantine of all the states in the Union. It would mean the destruction of all exposed and diseased hogs, and the thorough cleaning and disinfection of all contaminated premises, equipment, etc. There would be necessity for absolute restriction of the movement of swine from infected districts to clean

<sup>1</sup>Received for publication, December 24, 1923.

territory. It would call for the prohibition of swine importation from all countries known to have cholera.

When we call to mind the herculean task required to stamp out foot-and-mouth disease, in localized districts, we may have a fair conception of what would face us in an attempt to eradicate hog cholera, when we consider that there are very few sections in the country that are not more or less infected. Considering the fact that losses from hog cholera may be controlled through immunization, it would seem that the situation would not be improved by efforts at extirpation, since the wholesale slaughter of herds would disturb the progress of the swine industry to such an extent as to bring it practically to a standstill, for a time at least. It would have indirect serious results on other lines of farm endeavor, and would greatly curtail the supply of meats and meat-food products.

#### ARE THESE METHODS PRACTICAL

We have an anomalous situation in the eradication of infectious diseases of animals. Regarding foot-and-mouth disease, there has always been a determination to rid the United States of this plague, actuated by public demand. Regarding hog cholera, however, eradication is seldom considered. Swine owners are seemingly content to risk losses from year to year, rather than exert a proper effort in the direction of prevention, namely, immunization and the application of sanitary measures. Laws are lacking in many States to restrict the movement of sick and exposed hogs, to compel the proper disposal of dead animals, and to quarantine, clean, and disinfect dangerous premises. Where such laws now exist, too frequently they are not enforced.

What are the factors responsible for the restriction of efforts to eradicate hog cholera? Perhaps the chief one is the fact that the disease occurs, or rather has occurred in the past, as an epizootic at widely separated periods, usually every ten years. Between these periods, or waves, outbreaks of hog cholera have been sporadic and not sufficiently general to arouse the interest of entire communities or a combination of communities. In other words, a public demand has not been created.

England, for a number of years, directed its efforts to the eradication of hog cholera through the slaughter method. Even with the backing of laws, rules, and regulations rigidly applied, the enterprise proved a failure and was abandoned on the re-

commendation of a special committee appointed to look into the matter. It is a notable fact that losses from cholera have been no higher in England since the abandonment of the plan than while it was in force.

Public officials, both State and Federal, realizing the extent of infected territory and the cost in public funds of attempting effective quarantine, have subordinated their desire for the eradication of hog cholera to the antagonism that would inevitably result, on the part of farmers, swine growers and dealers, live stock associations, merchants, bankers, and legislators, to such a program. Since, in order to be successful, an attempt to eradicate cholera from swine herds would need the undivided support of these agencies, as well as that of state and municipal authorities, legislatures and Congress for laws and funds, the scope of the problem is apparent. And, in the full knowledge that such support is not to be expected, an attempt at eradication is considered to be futile and ill advised. In view of all these facts and the serious results that would inevitably follow, as already pointed out, is it a wise policy to urge the eradication of hog cholera, or even consider plans to that end?

#### WHAT IS THE TRUE SITUATION?

That the annual losses from hog cholera are still too high is not to be denied. Any loss of whatever extent is to be regretted. But it is quite within the scope of reasonable efforts greatly to decrease the number of hogs killed by cholera each year. With the use of the immunizing treatment (anti-hog cholera serum and hog cholera virus), it is possible to make hog raising a safe and profitable enterprise, provided these biologics are properly administered and certain instructions carried out regarding the care of herds following inoculation. Immunization of swine against cholera is not a theory. The use of serum alone, as a temporary protection, and of serum and virus to confer lasting immunity, was followed in laboratories for a considerable period before it was publicly announced as reliable. Even then, experiments were conducted in the field under farm conditions to determine the value of the treatment in actual practice. The results were highly satisfactory and proved that under correct methods of application a community may avoid losses from cholera through the use of the immunizing treatment.

It may be asked why swine immunization against hog cholera is being discredited in some sections and by some individual

hog men. It is a fact that some unfavorable results have occurred following the use of serum and virus, but in all such instances a thorough survey of the true conditions existing would have completely exonerated the principles involved in the action of these biologics in producing immunity under favorable conditions. First of all, these products must be active (the serum potent and the virus virulent) to have the desired results as immunizing agents. Next, the treatment must be given by a competent veterinarian or some layman with the proper amount of training and experience and able to recognize and differentiate one disease from another.

Animals must be free from disease when inoculated, although hogs in the very early stage of cholera usually recover and acquire a lasting immunity following treatment. The work must be done in a clean, aseptic manner, the necessary disinfectants being used to sterilize the instruments, the hands of the operator, and the seat of injection in the animal. Correct dosage of both the serum and virus is essential. The temperature of each hog treated should be taken, as the thermometer often reveals conditions that could not be observed through a casual examination. It should be remembered that hog cholera virus is the active element of the disease, and if handled carelessly may create new centers of infection. Haste or indifference on the part of the operator may lead to undesirable results, and in such instances he usually attempts to save his reputation at the expense of the serum and virus.

Often, too, the swine owner, whose hogs have not responded favorably to the immunizing treatment, is prone to lay the blame on anything or anybody but himself. As a matter of fact, the lack of attention on his part, in following the instructions given regarding the feeding of the animals and the proper care of them, is frequently responsible for unfavorable results. A child vaccinated for smallpox and immediately subjected to improper conditions of living will feel the reaction of the vaccine more acutely than the one who is kept quiet, not overfed, and not exposed to cold or wet weather. The same is true of the hog receiving the simultaneous treatment (serum and virus) against hog cholera. The herd should be kept on a light diet for a week or ten days following treatment. It should be warmly housed in cold weather. In summer, it should have the run of a pasture lot if such is available. The animals should be kept away from filthy wallow holes, should be fed on clean, sanitary

floors, and should have access to a plentiful supply of clean drinking water. The corn or other heavy ration should be restored gradually.

The high cost of producing anti-hog cholera serum has been a drawback to its general use. Conditions under which this biological product must be made, in order to be potent, sterile, and reliable, require the investment of considerable money. The serum is made from live hogs, which animals must be free from disease and kept under the best sanitary conditions. A large number of healthy pigs must be kept on hand on which the serum is tested for potency, under the supervision of Federal inspection, before the product is offered to the trade. A still larger number must be used in the production of virus. All these items in the process of serum production mean the installation of expensive equipment and, consequently, the cost of anti-hog cholera serum remains high. This feature and, in some instances, the unscrupulous demands of veterinary practitioners for their services, have led to a questionable phase of swine immunization, namely, the placing of the treatment in the hands of laymen in order to avoid the added expense of veterinary fees.

Unfortunately, too many veterinarians look upon the immunization of swine in the light of a remunerative service only. Too often the operation is performed on the basis of time consumed, instead of considering the necessary prophylaxis that should be followed in the administration of biological treatment. This fact is substantiated by the amount of unfavorable results observed, and it has been a contributing cause in the movement for farmer vaccination. The hurried visit of the veterinarian, his neglect to observe the conditions closely, his haste (and sometimes his carelessness) in the inoculation of the animals are faults less excusable because of his professional training and experience. And when, through his inattention to details, the herd reacts unfavorably to treatment, it is no wonder that the layman believes himself able, with a little instruction, to do the work creditably. We are thankful that but few of the practitioners in the country are responsible for such occurrences, but these few have caused a rather unwholesome situation, and have brought a considerable amount of criticism to the profession at large.

It is not denied that the intelligent farmer may learn to inject serum and virus into his own hogs or those of his neighbors, but the mere operation of a syringe is quite a minor factor in the

successful application of the treatment. One must be able to recognize the physical condition of swine before attempting to administer any treatment successfully. The use of serum and virus may be of no avail if the herd is affected with any form of disease, except, perhaps, hog cholera in its very early stage. Is the average layman qualified to differentiate between hog cholera, pneumonia, "flu," enteritis, tuberculosis, anthrax, and a multiplicity of other disease to which hogs are susceptible? Unless he has had extensive training in the observation of symptoms; unless he is adept in the use of the thermometer; unless he can autopsy a dead animal, recognize the various organs, and is able to understand the lesions produced by certain specific diseases, treatment by the layman, and particularly the immunizing treatment against hog cholera, which involves the use of a virulent biologic, is not for the best interest of the swine industry. And it can not be supposed that a seven-day course of instruction will impart all of the above knowledge.

#### WHAT IS THE ANSWER?

Since it has been shown that the eradication of hog cholera is not practical under existing conditions, there remains the problem of controlling outbreaks of the disease and cutting down the amount of losses. If old centers of infection can not be eliminated, extreme care should be taken to prevent the creation of new ones. Infection may reach a herd in a number of ways, some of which are as yet unexplainable. Suspicion still rests on several means of spreading infection, such as the roaming cholera hog, the roving dog, the purchase of infected or exposed stock, the visitor from infected to clean premises, the feeding of raw garbage in some instances, and the careless use of cholera virus. All of these are factors that swine growers can easily guard against without expense. But we shall always have with us the individual who absolutely refuses to be guided by sane advice, and who will continue to disregard the simple precautions necessary to avoid trouble, even at the risk of bringing to a community worry, inconvenience, and possible loss. There is only one remedy under such a circumstance, and that is to keep swine herds in the vicinity immunized against danger of cholera infection. It is known beyond a doubt that the simultaneous treatment (serum and virus) gives lasting protection against hog cholera. In the cases where the disease has reappeared in herds supposedly immunized, investigation has

shown that the unfavorable results were caused, for the most part, (1) by the use of impotent serum or a non-virulent or attenuated virus, (2) by under-dosage of either the serum or virus, (3) by faulty technique in the application of the treatment, and (4) last, but not least, the failure of the owner to follow instructions regarding the feeding and handling of the animals after immunization.

The adoption of sanitary measures in the hog lot, in the pen, and in the shelter is highly important. Whether the herd is immunized or not, it pays to have the hogs well kept, well housed, and free from internal and external parasites. The same system of sanitation that will prevent the introduction and propagation of disease germs and of the eggs of parasites will often prove effectual in preventing the introduction of hog cholera infection into a herd. If all the facts and the knowledge that have been learned regarding the nature of hog cholera were given proper recognition, and the known methods of prevention put into practical use generally, it is reasonable to believe that the disease would soon be brought to a status where it would no longer constitute a serious menace. But after all is said and done, we can not get away from the fact that anti-hog cholera serum has practically solved the problem of safety in the raising of swine.

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### PACKERS PAY PREMIUM ON PORK

The first shipment of hogs, from Illinois, to receive the premium of 10 cents per 100 pounds, which the packers have agreed to pay on hogs bred and fed within a tuberculosis-free county, was purchased by Wilson & Company, of Chicago, recently, at \$7.65 per cwt., the top for the day. The ten-cent premium, over and above the selling price, was paid by a separate check. The hogs were sold by Mr. Everett C. Brown, president of the Chicago Live Stock Exchange.

Edgar County was the first county in Illinois to become "modified accredited area," under the area plan inaugurated about three years ago. It is said that the now-famous tragedy of the Kelly family, in which one tuberculous cow infected five of the seven Kelly children with tuberculosis, is largely responsible for the impetus given tuberculosis eradication work in this vicinity. Forty-six counties in Illinois are now engaged in systematic testing in the hope of becoming tuberculosis-free.

# A STUDY OF THE ENZYMIC ACTION OF EXTRACTS OF THE DUODENAL GLAND REGIONS OF THE DOMESTIC ANIMALS

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## INTRODUCTION

The duodenal glands were first described by Brunner, in 1686, though it is said that they were discovered by Wepfer, nine years earlier. Concerning the physiology of these glands, very little of an exact nature is known. Modern textbooks of physiology contain little or no information in this regard. A review of the literature shows that comparatively little work has been done on the physiology of these glands, such results as have been reported being in a number of cases contradictory. In herbivorous and omnivorous animals, the duodenal glands are found extending for a considerable distance immediately behind the pylorus. In the horse, for example, they occupy a zone of six to eight meters, this being about one-third the entire length of the small intestine. In the omnivorous pig, the zone extends about four meters; however, in man, it is only about twelve to fifteen centimeters in length.

Upon first thought, it might seem strange that the knowledge of the physiology of these glands, occupying as they do such extensive zones, especially in herbivora, is so obscure, but when their anatomical relation to the intestinal glands is recalled, there is little wonder that the knowledge of their function is so incomplete, and that results in the hands of different investigators have been in many cases contradictory. This investigation was undertaken for the purpose of determining what, if any, enzymes are produced by the duodenal glands in the horse, the ox, the sheep, and the pig.

## THE HISTOLOGY

The histology of the duodenal glands and their distribution in different species are somewhat suggestive as to their function. Writers at various times have advanced hypotheses concerning their physiology, based upon purely anatomical and histological facts. On the one hand, some investigators, because of the histological similarity between these glands and those of the

pyloric gland zone of the stomach, have held that the two groups of glands are functionally equivalent; that is, that the duodenal glands produce an enzyme proteolytic in nature.

Murphey,<sup>1</sup> on the contrary, has for years taught that the duodenal glands in herbivora probably produce an amylolytic enzyme. This assumption he bases on the following facts: In most herbivora and omnivora the duodenal gland zone is long; in carnivora, short; in the stomach of the pig, as high up as the fundus, are found occasional islands of duodenal-like glands; Pavlov<sup>2</sup> reports that the duodenal glands secrete an amylolytic enzyme; Bengen and Haane<sup>3</sup> state that an amylolytic enzyme is produced in the stomach of the pig; the pig under domestication is largely herbivorous, and this species has a long duodenal gland zone (about four meters); with the most exacting histological technic, Murphey has never been able to obtain a mucous reaction in the duodenal glands.

#### TYPES OF GLANDS

The duodenal (Brunner's) glands (*glandulae duodenales*) are tubulo-alveolar glands, whereas the intestinal (Lieberkuhn's) glands are of the simple, tubular type. Histologically they are considered by some to represent a continuation into the intestine of the pyloric glands, although the duodenal glands are sharply distinguished from the latter by their larger size. According to Schiefferdecker,<sup>14</sup> the pyloric glands and the duodenal glands are identical in man, the pig, the dog, and the cat. He places the pyloric and the duodenal glands in one group and terms them the glands of the pyloric zone. Bogomoletz<sup>3</sup> does not agree with Schiefferdecker, stating that the duodenal glands do not appear as a continuation of the tubular glands of the pyloric region. According to Ellenberger, the duodenal glands occupy in the horse a zone of 6 to 8 meters behind the pylorus; in the ass, 5 meters; in the ox, 4 to 5 meters; and in the sheep, 60 to 70 centimeters (two feet or more, Sisson); in the goat, 20 to 30 centimeters; in the pig, 3 to 5 meters; in the dog, 1.5 to 2 centimeters; in the cat, 2 to 5 centimeters; and in man, 12 to 15 centimeters.

The secreting portion of the duodenal glands is found principally in the submucosa, though in some cases also in the deeper parts of the mucous membrane. The ducts of the glands open on the free surface between the villi by means of crypt-like tubules which are lined by tall, columnar epithelium and

can only with difficulty be distinguished from the adjacent intestinal glands; or in some cases they open into the fundus of the intestinal glands. The ducts branch and pursue a somewhat tortuous course to the fundus of the gland, where the terminal alveoli of each subdivision of a duct are found. The secreting epithelium of the duodenal glands consists of tall, columnar cells similar to those lining the ducts. The cells contain large nuclei, surround a large lumen, and rest upon a cellular (Jordan and Ferguson) basement membrane. According to others (Lewis and Stohr), the basement membrane is structureless. Secretory capillaries extending out from the lumen between the cells have been noted. When loaded with secretion, the cells are swollen and clear, but after a period of activity they become shrunken and granular. The nucleus becomes progressively flattened as the cell fills with secretion.

#### THE LITERATURE

Middeldorpf<sup>4</sup> reports an experiment upon the digestive activity of the duodenal glands. Protein was not digested. Starch was split into sugar. Mendeldorp<sup>5</sup> also found a diastatic enzyme in the extract of the gland substance. Grutzner<sup>6</sup> states that extracts of the duodenal glands contain a diastatic enzyme. Landois and Stirling<sup>7</sup> make the following statements: "A watery extract of the glands causes: (1) Solution of proteins at the temperature of the body (Krolow). (2) It also has a diastatic (?) action. It does not appear to act upon fats."

Bogomoletz<sup>8</sup> summarizes the confusion existing as to the function of the duodenal glands when he says that some regard these glands as mucous glands, others as secreting an enzyme of the nature of pepsin, and still others (Budge and Krolow), as secreting a diastatic enzyme. He states that the duodenal glands are alveolar in type, and do not appear as a continuation of the tubular glands of the pylorus. From feeding experiments conducted upon rabbits, followed by histological examination of sections of the duodenal glands of these animals, he found that protein feeding greatly increased the amount of zymogen granules in the gland cells, fat feeding caused a decrease, and feeding with cane sugar caused greater mucus production. He does not report results from starch feeding.

According to Krolow,<sup>9</sup> the fluid secreted by these glands has the power of converting starch into sugar, and completely dissolves raw fibrin, but has no effect upon fat and coagulated

albumin. Glaessner,<sup>10</sup> by scraping off the mucous membrane of the duodenum after the piece had been thrown into boiling water for two minutes, reports the finding of a proteolytic enzyme ("pseudopepsin") in the extracts of the submucous glands, and an amylolytic enzyme in the extracts of the glands of the mucous membrane. He used the pig and the dog, and reports a total of only four experiments.

Scheunert and Grimmer<sup>11</sup> found no proteolytic enzyme (Glaessner's "pseudopepsin"), and no rennin in the duodenal glands of the horse, ox, and pig, but state that an amylolytic enzyme is produced by the duodenal glands of these animals. They used pressed juices of the gland substance. Pavlov<sup>2</sup> states that the juice secreted by the duodenal glands unquestionably contains a diastatic enzyme, and that a small amount of ptyalin seems to be present both in the intestinal juice and in extracts of the mucous membrane. Gachet and Pachon,<sup>12</sup> in their experiment, in which they introduced cylinders of coagulated ovalbumin into the duodenum of the dog, did not exclude the possibility of the action of pancreatic juice on the introduced protein.

#### PONOMAREV'S EXPERIMENT

Ponomarev<sup>13</sup> carried out an experiment at the proposal and under the guidance of Pavlov, to determine the properties of the juice of the duodenal glands. Two dogs were used, and, by a very delicate operation described, he succeeded in establishing permanent duodenal gland fistulae in these animals. The secretion of the juice was continuous. It was colorless, thick and thread-like, and with the admixture of grayish lumps of mucus. Upon standing, the mucus precipitated, and the juice became clear. The reaction was always alkaline. The juice showed no action on protein in alkaline medium but, using the method of Mett, he reports digestion of fibrin and of serum albumin when the juice was acidified. He considers the finding of an enzyme, similar in action to pepsin, logical in view of the analogy between the pyloric glands and the duodenal glands, and even the existence of a transitory belt between them, as pointed out by Schiefferdecker<sup>14</sup> and others.

It should be noted that Ponomarev's work was done on the dog only, which species possesses a very short duodenal gland zone (1 to 2 cm.). It would be hazardous to apply his findings to the herbivorous horse, ox, or sheep, or to the omnivorous pig, with their long zones of duodenal glands. In addition, it

is agreed with Ellenberger and Scheunert<sup>15</sup> that there is a possibility of a fistulous secretion from the duodenal glands of the dog being mixed with the secretion of the neighboring pyloric glands, in view of the very short duodenal gland zone of this animal.

Villemin,<sup>16</sup> from purely histological and anatomical facts, advanced the hypothesis that the duodenal glands of the horse supplement the relatively small gastric gland zone, and thus secrete a proteolytic enzyme. Costa,<sup>17</sup> working on the horse, specially prepared the intestinal and duodenal glands by extraction with glycerin. Using these extracts in digestive experiments, he found that the glands of Brunner possess strongly the power of converting starch into sugar, but have no effect on albumin or fat, thus confirming Krolow's observations; and that the extract of Lieberkuhn's glands of the small intestine immediately changes starch into sugar. He further found that an extract of the glands of the large intestine is without effect upon starch, albumin, and fats.

In the calf, ox, horse, sheep, and fowl, Fisher and Neibel<sup>18</sup> found an amylolytic enzyme in extracts of the small intestinal mucous membrane. Pregl<sup>19</sup> investigated the juice from a Vella fistula of the small intestine of a lamb, and starch as well as glycogen was digested with the formation of dextrose. Ellenberger and Hofmeister<sup>20</sup> extracted the fresh and dried intestinal mucous membrane of the horse with carbolated water and other agents, and found in all regions of the intestine an amylolytic enzyme.

#### EXPERIMENTAL METHODS

Since it is impossible to obtain the secretion of the duodenal glands in pure form, by either Thiry's fistula or Vella's modification of Thiry's method, and since the establishing of an exclusive duodenal gland fistula seemed to the writers to be entirely out of the question (except perhaps in the dog, Ponomarev claiming to have succeeded in this animal), several methods to free the duodenal submucosa from the mucous membrane were tried in order that by extraction any enzymes in the glands of the submucosa might be obtained free from those of the mucosa. Attempts were made to obtain at least the deeper portions of the submucosa by working from without inward, having filled the lumen of the piece of intestine with physiological salt solution. This method proved to be entirely impracticable, owing to thinness of the

submucosa, and the consequent ease with which it is perforated.

It was then decided that an attempt should be made to isolate the submucosa by removing the mucous membrane, thus leaving the former free of intestinal glands, these being confined to the mucous membrane. By inverting the piece of intestine and filling it with melted paraffin, it was found that the mucous membrane could apparently be removed satisfactorily, the paraffin, after solidification, offering sufficient resistance to enable this operation to be done. The objection to paraffin is its fairly high melting-point ( $50-57^{\circ}$  C.), and the possibility of this temperature proving injurious to any enzymes present. It was therefore discarded in favor of cacao butter (oil of theobroma, U. S. P.), a fixed oil, solid at room temperature, melting at  $30-35^{\circ}$  C., a temperature below that which is injurious to enzymes.

#### THE AUTHORS' METHOD

The details of the method adopted by the writers for freeing the submucosa of mucous membrane are as follows: The desired pieces of intestine (except those from the horse and the dog) were obtained at the College abattoir shortly after the animals were slaughtered for food. The pieces were then brought to the laboratory, where they were freed of ingesta and washed in running water for variable lengths of time (several minutes to sixteen hours). The individual pieces of intestine were then freed of serous membrane and as much of the muscular coat as could conveniently be removed. Next, the segment of intestine was turned inside out, one end closed with a clamp, and the whole piece filled with sterile, melted cacao butter, at or near the temperature of the body. The other end was clamped, and the filled piece hung in the refrigerator to allow the cacao butter to solidify. If it was not practicable to work up a piece completely as soon as the cacao butter hardened, the piece was usually covered with a damp sterile towel and allowed to remain in the refrigerator for variable lengths of time, sometimes until the next day.

The mucous membrane was removed from the segment of intestine by scraping with a sharp knife, a curved bistoury being admirably adapted to this purpose. The piece was always thoroughly scraped, so as to make sure that the submucosa was as clean as possible. In the case of the horse, which species has a thick and resistant mucous membrane, especially in the duodenum, it was possible in that location to dissect the mucous

membrane actually from the underlying submucosa, thus being, it seems to the writers, absolutely certain that the latter was not contaminated with mucous membrane.

The submucosa, or the mucous membrane, or both, were separately extracted with glycerol, the time allowed for the extraction being quite variable. (See table I.) Apparently this time element was not important. Watery extracts were occasionally used, as is shown in the tables. In many cases, parts of the intestine other than the duodenal gland zone were extracted for purposes of deduction, control, etc. The extracts were tested for their digestive action on starch, protein, and fat, in alkaline, neutral, and acid solutions.

#### EXPERIMENTS TO DETERMINE DIGESTIVE ACTION OF EXTRACTS ON STARCH

The starch used in all cases was in the form of a one per cent boiled paste, which showed no reducing sugar when tested with Fehling's solution. Five cubic centimeters of this paste in each of three test tubes were mixed with equal amounts of a one per cent solution of sodium carbonate, distilled water, and 0.2% hydrochloric acid, respectively. To each of these tubes was added 1.0 cc of the extract to be studied for enzymic action. Thus, the first tube contained 0.5% starch in 0.5% sodium carbonate solution, the second 0.5% starch in water, and the third 0.5% starch in 0.1% hydrochloric acid, with 1.0 cc of extract in each case. Control tubes were identical with the experimental ones, except that the extract was either omitted or boiled. The usual period of incubation for the starch-extract mixtures was 48 hours, though there were variations as is noted in the tables. The temperature was  $37\frac{1}{2}^{\circ}\text{C}$ . At the end of the incubation period, the experimental and control tubes were tested for the presence of reducing sugar, using Fehling's solution, which a previous test had shown to be reliable. No quantitative determinations were made, as these would have limited value owing to the fact that it was not possible to control the concentration of enzyme and the amount of tissue fluid originally present in the specimens extracted. Control tubes in several cases reduced Fehling's solution, but in all of these cases the reduction was so slight, when compared to the experimental tubes, as to appear entirely negligible.

No extract (out of a total of 63 tested) from any part of the intestine of any animal studied showed digestive action on

TABLE I—AMYLOLYTIC ACTION OF EXTRACTS FROM DIFFERENT REGIONS OF THE INTESTINE OF DOMESTIC ANIMALS

Species	Kind of extract	No. days tissue was extracted	Ratio of extract to medium	Digestion in hours	A Mucous mem- brane duodenal gland zone	B Submucosa duo- denal gland zone	C Mucous mem- brane ileum	D Submucosa ileum	E Mucous mem- brane colon	F Submucosa colon
Ox I	Glyc.	10	1:5	46		++				
Ox II	"	"	"	44 24	++	++				
Ox III	"	A 18 B 12 C&D 13	"	48	a +	+++	—	+		
Ox IV	"	11	"	"	+	+++				
Ox V	"	A 14 B 13	"	"	+	++				
Ox VI	"	14	"	"		+++				
Ox VII	"	7	"	"			—	+		
Ox VIII	"	"	"	"			+	+		
Horse I	"	—	2:5	24		+++				
Horse II	"	—	"	"		++				
Horse III	Watery	Few hrs.	2:5	24		++				
Horse IV	Glyc.	9	A 1:5 B 3:5	A 48 B 44	+	++				
Horse V	"	"	1:5	48		+	+	+		
Horse VI	"	11	"	"	+	+	++	++	+	+
Pig I	"	"	"	46		++				
Pig II	"	5	"	24		++				
Pig III	"	7	"	B&C 48 D 44		+	+++	++		
Pig IV	"	"	"	48		+++ <sup>a</sup>				
Pig V	"	14	"	"		++				
Pig VI	"	8	"	"		++ <sup>a</sup>				
Pig VII <sup>b</sup>	"	2	"	"	+++ <sup>a</sup>	++	+	++	+++ <sup>a</sup>	++
Pig VIII	"	"	"	"	+++	++	+++	++	++	++
Sheep I	"	11	"	"		+++				
Sheep II	"	19	"	"	a +	a ++				
Dog I <sup>c</sup>	Watery	½ Hr.	2:5	16	d ++	d +++				

a+ in alkaline solution also.

b Each segment of intestine washed in running water for 16 hours before extraction.

c Each segment of intestine washed in running water for 6 hours before extraction.

d Section of duodenum posterior to duodenal gland zone.

starch in the acid solution used, 8 out of 63 showed digestion in the alkaline solution, and 63 out of 65 in neutral.

Table I shows the kind of extract used, the ratio of extract to medium, the duration of incubation in hours, and the results of digestion in neutral medium. Footnotes show where there was digestion in alkaline medium. The plus signs should be interpreted as follows: +, slight reduction of Fehling's solution; ++, medium reduction; and + + +, strong reduction, as determined simply by inspection of the precipitate of cuprous oxide.

Table II is similar to table I, except that the vascular systems of the animals from which the pieces of intestine were obtained were irrigated with water for several hours immediately after bleeding in an attempt to free the tissues of blood and lymph, and thereby any enzymes contained in these fluids. The pieces of intestine were then obtained and treated in the usual way.

TABLE II—AMYLOLYTIC ACTION OF EXTRACTS FROM DIFFERENT REGIONS OF THE INTESTINE OF OX AND OF DOG WHOSE VASCULAR SYSTEMS HAD PREVIOUSLY BEEN PERFUSED WITH WATER

Species	Kind of extract	No. days tissue was extracted	Ratio of extract to medium	Digestion in hours	A	B	C	D	E	F
					Mucous membrane duodenal gland zone	Submucosa duodenal gland zone	Mucous membrane ileum	Submucosa ileum	Mucous membrane colon	Submucosa colon
Ox IX	Glyc.	2	1:5	48	++	++	+	++	+	+++
Dog II	Watery	18 hrs.	"	21	+	+	- <sup>a</sup>	+ <sup>a</sup>		

<sup>a</sup> Segment of duodenum posterior to duodenal gland zone.

#### EXPERIMENTS TO DETERMINE DIGESTIVE ACTION OF EXTRACTS ON PROTEIN

Three kinds of native protein were used: shreds of boiled beef muscle, coagulated egg albumin in Mett's tubes, and heat-coagulated blood proteins in Mett's tubes.

In preparing the Mett's tubes containing egg albumin, white of egg was sucked into sterile Mett's tubes and these were placed in water at 95° C. for about five minutes and when ready for use were cut into proper lengths.

For the preparation of Mett's tubes with blood proteins, plasma from oxalated horse blood was drawn into the sterile tubes and coagulated by heating in water at 95°C. for about

ten minutes, and when ready for use the tubes were cut into proper lengths.

Action of the extracts on protein was tested in 1% sodium carbonate solution, distilled water, and 0.2% hydrochloric acid solution. The ratio of extract to digestive medium was 1:5. Either chloroform or toluol was in nearly all cases used to prevent bacterial action. Control tubes contained the medium and the protein without the extract.

Out of the tests of the action of a large number of extracts from the mucous membrane and the submucosa of the different regions of the intestine of the several species of domestic animals, digestion of protein was found in only two cases. These two extracts were from the duodenal gland substance of the ox, the action was in acid medium only, and the material digested was blood proteins in Mett's tubes, chloroform being used to prevent bacterial action. The duration of the incubation was seven days at 37.5°C.

Extracts of the following portions of the intestine of the species named showed negative results on coagulated blood proteins in Mett's tubes in alkaline, neutral, and acid solution, the duration of incubation being one week at 37.5°C.: Mucous membrane of duodenal gland zone of ox (two animals), of horse, and of pig (two animals); submucosa of duodenal gland zone of ox (three animals), of horse (two animals), of pig (five animals), and of sheep; mucous membrane of ileum of ox (two animals) and of pig (three animals); submucosa of ileum of pig (two animals); mucous membrane of colon of pig; submucosa of colon of pig (two animals).

Extracts of the parts named below showed negative results on blood proteins and on egg albumin, both in Mett's tubes. The duration of the incubation in case of the blood proteins was one week, and in case of the egg albumin 40 hours, at 37.5°C. Antiseptic was used. The following were the extracts tested: mucous membrane of duodenal gland zone of horse and sheep and submucosa of duodenal gland zone of sheep, pig, horse, and ox.

Extracts of the following parts of the intestine showed negative results on shred of boiled beef muscle, the duration of the incubation, for the most part at room temperature, being 13 days and no antiseptic was used: mucous membrane of duodenal gland zone of ox; submucosa of duodenal gland zone of pig (two ani-

mals), of horse (three animals); and submucosa of ileum of pig.

#### EXPERIMENTS TO DETERMINE DIGESTIVE ACTION OF EXTRACTS ON FATS

Extracts of the following named parts of the intestine did not hydrolyze fresh butter in alkaline or neutral solution, in controlled experiments, using phenolphthalein and methyl orange, respectively, as indicators: mucous membrane of duodenal gland zone of ox; submucosa of duodenal gland zone of ox, of horse (two animals), and of pig; and submucosa of ileum of pig.

Extracts of duodenal gland zone submucosa of horse (three animals) and of ox did not hydrolyze olive oil in experiments in which the amount of acid in 48-hour incubated experimental and control tubes was titrated against N/20 sodium hydroxide, using phenolphthalein as an indicator.

#### DISCUSSION OF RESULTS

*Action of extracts on starch.* It is emphasized that out of a total of 65 intestinal extracts from five species, only three failed to digest starch in neutral solution. This is remarkable when it is considered that many different portions of intestine, both mucous membrane and submucosa, were extracted and tested. Even extracts of submucosa of ileum and colon, which it is generally conceded are glandless, showed great powers of digesting starch. Included in these 65 extracts were ten from various parts of the intestinal tracts of an ox and a dog whose vascular systems had been perfused with water for a number of hours before obtaining the tissues for extraction. Only one of these ten extracts lacked amylolytic powers (see below and table II).

Though quantitative estimations of the amounts of reducing sugar formed were not made for reasons already given, yet extracts of the submucosa of the duodenal gland zone showed in most cases greater power of splitting starch than extracts of other regions of the intestine. It is admitted, however, that a larger number of extracts were prepared from this region than from any other, so that such a comparison would perhaps not be entirely accurate. It is clear, however, that extracts of the submucosa of the duodenal gland zone possess almost without exception marked amylolytic powers, and in no case was this power absent.

The wide distribution of an amylolytic enzyme in the wall of the intestine, irrespective of the species, or of the location of

the part studied, or of whether it contained glands, made the writers wonder whether or not this amylase was present as a product of the cells of the tissue extracted, or simply owed its presence to the fact that the structure contained blood and lymph. Accordingly, the vascular systems of two animals were perfused thoroughly for hours with water, yet extracts of the intestines of the animals so irrigated still showed the presence of a widely distributed amylolytic body (see table II). If the enzyme in question is present simply in virtue of the presence of blood and lymph, this shows how difficult or impossible it is to free an organ or tissue completely of tissue fluid.

To throw further light on the distribution of this enzyme, extracts of the following tissues or organs were made: skeletal muscle, mesenteric lymph gland, epidermis and derma, and subcutaneous tissue, all from the pig; and mucous membrane, and muscular coat with attached submucosa, of the rumen of the ox. These extracts without exception possessed powerful amylolytic properties, one per cent starch paste being used. Furthermore, the observations of numerous investigators that the blood has amylolytic powers were confirmed. The animals used were the horse and the dog. The amount of blood amylase is regarded by Mathews<sup>21</sup> as being small.

*Action of extracts on protein.* It is likely that the acid protease demonstrated in extracts from the submucosa of the duodenal gland zone of two cows was pepsin from the stomach. No other extracts from any part of the intestine of any species, out of a large number of extracts tested, were active on native protein.

*Action of extract on fats.* No lipase was found in any case.

#### SUMMARY

- (a) The lack of agreement among investigators as to the functions of the duodenal glands is noted.
- (b) The purposes of this investigation are given.
- (c) The histology of the duodenal glands is briefly compiled.
- (d) A brief review of the available literature on the functions of the duodenal glands is included.
- (e) An original method of freeing the submucosa of the intestine from the mucous membrane is described.
- (f) Experiments are cited in which the digestive actions of extracts of the duodenal gland substance and many other parts of the intestine of the domestic animals were tested on starch, protein, and fat. Two tables are given.

(g) Extracts of a number of tissues other than those of the intestine were tested for amylolytic action.

#### CONCLUSIONS

##### 1

(a) Comparatively few investigators have studied the functions of the duodenal glands.

(b) The secretion of these glands has never been obtained in pure form, so far as the writers were able to find, except perhaps from two dogs by Ponomarev. His methods and findings are questioned for the reasons given in the text.

(c) Such results as have been reported from the use of extracts of the duodenal glands are in a number of cases contradictory.

##### 2

(a) In this work, extracts of the duodenal gland substance of the horse, ox, sheep, and pig were found invariably to contain an amylolytic enzyme active in neutral solution. In very few cases was the enzyme active in 0.5% sodium carbonate solution and in no case in 0.1% hydrochloric acid solution.

(b) Extracts of mucous membrane and submucosa of other parts of intestine in nearly all cases showed amylolytic powers.

(c) This amylase was still present in considerable amounts in the several parts of intestine of two animals (ox and dog) whose vascular systems had been perfused with water for several hours.

(d) The presence of an amylase in blood and lymph has long been recognized. This was confirmed by the writers for the blood of the horse and the dog.

(e) Extracts of many tissues contain an amylolytic enzyme.

(f) Whether the amylase in the duodenal gland extracts is produced there by the gland cells, or is simply present as a result of the presence of the blood and lymph, or both, the writers are not prepared to state. In general, the amylolytic action of duodenal gland substance extracts was greater than the action of extracts of the submucosa of other regions, which leads to the belief that the duodenal gland cells themselves produce an amylase.

(g) The unqualified statement by some investigators, using extracts of the duodenal gland substance, that an amylase is produced by the duodenal glands, is not justified in the light of these findings that extracts of the mucous membrane or submucosa of any part of the intestine, or of numerous other tissues, contain an amylolytic enzyme.

(h) The acid protease found in the duodenal gland extracts of two cows was likely pepsin from the stomach.

(i) In general, the findings of some investigators, that extracts of the duodenal glands are proteolytic, could not be confirmed.

(j) Extracts of duodenal gland substance are not lipoclastic.

(k) The difficulty of freeing the duodenal glands from intestinal glands has, in a measure at least, been met. However, the difficulty of freeing the tissue of tissue fluid was apparently not overcome.

(l) Exact data as to the enzymes produced in glands are difficult to obtain by the use of extracts of the gland.

(m) Until the duodenal gland secretion is obtained in pure form, the question of the exact function of these glands cannot be conclusively settled.

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#### NEWS NOTES

A mysterious ailment is said to have caused the death of thirty horses in one barn during the past eight years, on a farm near Hopkinton, Iowa. Veterinarians are at a loss to account for the deaths of the horses, which have invariably been quite sudden.

In the eleven weeks ending March 16, 1924, there were 1071 outbreaks of foot-and-mouth disease in Great Britain. Since the present series of outbreaks began, in August, 180,832 animals have been slaughtered, over one-half of which were cattle.

## NUTRITION TRAILS<sup>1</sup>

By PIERRE A. FISH, *Ithaca, New York*

The influence of sunlight as a fundamental element in nutrition has not been adequately appreciated in the various attempts to solve Nature's policy in sustaining plant and animal life. Without sunlight there would be no plant life worthy of the name. Without plant life, there would be no animal life.

Aside from photography the chemistry of light is little understood. The influence of the active energy of the sun's rays upon the chemical changes going on in the plant is practically unknown. The plant, with its chlorophyll, is able to convert carbon dioxide and water, which of themselves possess no nutritive value, into sugar and starch which are valuable nutrients. The plant can convert ammonium nitrate, with other material from the soil, into protein, but Nature still holds the key to the secret. Plants do not show the same constructive ability in relation to the production of fat. Although vegetable oils are numerous and important, the evidence indicates that the plant has the power of transforming some of its own carbohydrates into this form of fat, rather than constructing it directly from basic material derived from the soil.

Important as are proteins, carbohydrates, fats and other organic compounds, the mineral or inorganic constituents are also essential to life. Sodium, potassium, calcium, magnesium, iron, phosphorus, sulphur and chlorin are found in the tissues and fluids of all living things; fluorin, silicon and iodine have been recognized in many plants and animals but only in certain tissues; copper, manganese, zinc and bromine have been found in marine invertebrates and manganese and zinc are not infrequently found in plants. The inorganic constituents function to a certain extent in the processes of diffusion and osmosis and in directing the metabolism or tissue changes of the organism.

In recent years another function of vital importance has been discovered in plants, and that is their power to construct vitamins. It is now known that these substances are absolutely indispensable to life, and plants alone can produce them. The animal body does not possess this function, although it may store the vitamins for a time in certain of its tissues and even pass

<sup>1</sup>Read before the Veterinary Medical Association of New York City, December 5, 1923.

them on to its offspring through its milk. The chemistry of vitamins is still too meager to offer any information concerning their production, but recent investigations indicate that sunlight plays an important part in their construction. One exception, however, should be noted in the case of the vitamin found in sprouting oats or wheat, where the sprouts obtained by germination in darkness are apparently just as efficient as those obtained in sunlight.

In a material sense the words of the Psalmist: "All flesh is grass," is true. Without the vitamins produced in plants, there could not be life or growth in animals. Herbivorous animals represent vegetable material transformed into flesh, and meat consumed from this source may, in this sense, be regarded as the modified product of plant life.

#### LIGHT PLAYS AN IMPORTANT RÔLE

Back of it all is light. The energy of life is derived from the energy of the sun. If any form of idolatry could be justified, that of the Sun-worshippers is the most worthy. Light has made its impress upon therapeutics. Its beneficial effects in the form of X-rays and ultra-violet rays have already been demonstrated and we are still, doubtless, only on the threshold of the knowledge of its chemical reactions with the tissues. Even in its destructive form, light is beneficial to humanity in that it destroys those organisms which work in darkness and which menace health and safety. Light is Nature's antiseptic and purifier.

Although much has been accomplished, the approaches to the problems of nutrition cannot yet be regarded as having reached the avenue stage with gentle grade and smoothly paved surfaces. They must still be looked upon as trails leading over rough and rugged territory with side and branching trails which lead out into the unknown.

The normal tissues of the animal body must be replenished by raw material from the outside world. In its raw form it cannot reach its destination. It must undergo a process of demolition, through the action of the digestive juices, in order to penetrate the barrier of the wall of the digestive canal, before it can be truly within the body, where it can be circulated to the tissues that can utilize it. The architecture of the food material must be considerably changed in order that the material may be built up into a different style of architecture in the tissues. In this

process much of the material is discarded and passed from the body as waste matter.

*The protein trail.* All protein must be broken down into its units, the amino-acids. The demolition must be complete in order that these units may be built up into animal tissues or utilized by the tissues for various purposes. Some of the units may be converted into carbohydrates and it is not impossible that some may be transformed into fat, although this is a matter that is not yet beyond the realm of controversy. The same amino-acid units are not found in all proteins. No single protein contains all of the amino-acids. Like the vitamins, certain of the amino-acids are essential for maintenance and growth. The problem in nutrition is to supply the proteins which contain the amino-acid units that are necessary for the proper functioning of the tissues.

*The carbohydrate trail.* The utilization of the carbohydrates in the food does not demand such complete demolition of their structure by the digestive juices as in the case of protein. The complex starch molecule is reduced to the sugar (dextrose) molecule which is still complex but simplified enough to be absorbed and used by the tissues—any excess being converted into animal starch (glycogen), and later reconverted into sugar when needed. That carbohydrates can be converted into fat is now an accepted fact, but the method by which the tissues are able to bring about this conversion has not yet been clearly established.

*The fat trail.* Fat, like the carbohydrates, does not undergo complete disintegration. Its structure is separated into the constituents—fatty acids and glycerin—each of which is capable of undergoing further changes. This separation occurs for the purpose of absorption. The constituents, for the most part, are immediately reunited as neutral fat in the form of fine particles commonly known as the “molecular basis of chyle.” These are collected in the lacteals and transferred through the thoracic duct into the blood circulation. The presence of minute particles in the blood has been known for some time. They have been referred to as blood dust or hemaconia and have been supposed to represent the debris or disintegrated particles of the various blood elements. S. H. Gage has shown that this view is erroneous. Using the dark-field microscope, which shows the particles with great clearness, he has shown that with a strictly protein diet (white of eggs) or a strictly carbohydrate diet (rice and sugar) or

a combination of the two diets, there was no material increase in the number of particles in the blood; but with a diet of a digestible fat (butter, cream, cod-liver oil, olive oil) there was soon a very decided increase in the number of blood particles. Since these particles take their origin from the chyle, he has applied to them the appropriate term of chylomicrons. The maximum number of particles are found in the blood in from three to six hours after the fat has been consumed, after which the number at first diminishes quite rapidly and then somewhat more slowly until there is a return to the original number. There is produced a temporary lipemia which is normal to the digestive processes. The rate of fat digestion will depend upon the amount and digestibility of the fat and upon the digestive power of the individual.

Our experiments show, in general, a similar curve for fat digestion in man, dog and cat. In the cow a different type of curve was obtained. The curve showed peaks of digestion alternating with periods of quiescence throughout the 24 hours. The result would indicate that the fat (butter) passed to the first three compartments of the stomach, some of it passing early into the abomasum and intestine, while the remainder was passed onward in instalments at later intervals.

In the horse, variable results occurred. A curve, similar to that of man and the carnivora, has been obtained, and again a curve in which a high chylomicron count has been found (after the horse had been fasted) at the initial or zero hour, just before the fat has been administered. The subsequent curve showed irregular fluctuations during the 24-hour period. This surprising result has occurred more than once and requires further investigation.

Experiments upon poultry gave, also, conflicting results. With some non-laying hens and a rooster the dark-field microscope showed scarcely any fat absorption in the 24-hour period insofar as the presence of the chylomicrons in the blood was concerned; yet we do have evidence, from another method, that there is a satisfactory fat absorption in the hen, at least during the laying period.

*Experiments with Sudanized fats.* Sudan III is a red dye which is soluble in fats and fat solvents. It clings tenaciously to the fat and even when the fat is separated into its component parts—fatty acids and glycerin—it still adheres to the fatty acids and when these are resynthesized into neutral fat, a pink or red

color is still in evidence. The red trail thus established makes it relatively easy to follow the course of the fat to its destination. If a series of animals be killed at proper periods during fat absorption and assimilation it is not difficult to demonstrate: (1) the pink chyle, in the engorged lacteals and thoracic duct; (2) by drying some of the serum of the blood and treating it with ether, an ether-extract showing a pink color can be obtained; (3) the fat deposits, or adipose tissue, will show a pink color, or if the color is weak some of the body fat may be treated as the blood serum and a pink ether extract obtained. The pink body fat may be made into soap of the same color; (4) the colored fat can be observed in the milk of some lactating animals, *e.g.*, goat, cat, rat, and probably others, but thus far negative results have been obtained from the cow. The pink color is, of course, confined to the fat constituent of the milk, but as the cream rises the color is more distinct and is found only in the cream layer. Pink milk has been observed in the stomach of the nursing offspring; (5) in laying hens fed with Sudanized fat, concentric rings of a pink color will appear in the yolk.

From these experiments we may conclude that some of the fat of the food is used in the production of some of the fat of the milk and, similarly, that some of the food fat contributes to the formation of the fat of the yolk of the egg. It is also shown that substances not commonly found in ordinary fats, Sudan III, can be deposited in the fat reserves of the body. It is reported that erucic acid, a constituent of rape oil, when fed to a dog, appears in its fat and that cows fed on maize oil yield butter of a low melting point.

The trail by which carbohydrates are converted into fat has not yet been sufficiently cleared to explain the details.

*The mineral trail.* The proximate principles, or organic foods, alone will not support life. There must be inorganic material for the maintenance and replenishment of the solid structures and for the proper concentration of the blood, lymph secretions and tissue juices. The red corpuscles must have iron; the bones, calcium. Sodium, potassium, magnesium, phosphorus and sulphur likewise play their part in the metabolism of the body and must be furnished with as much exactitude as the organic constituents.

*The endocrine trail.* The processes of nutrition occur in an orderly manner through metabolism—chemical changes which regulate the production of waste material in the tissues and its

replacement by new substance derived from the food. Metabolism is to nutrition what the mechanism is to a watch—it keeps things going. Any disturbance of metabolism, or of nutrition, causes effects which are more or less profound. The amount of evidence is increasing that there is a close relationship existing between certain, if not all, of the endocrine glands and the processes of nutrition. The removal of certain of these glands is attended by fatal results. An abnormal condition, according to the gland affected, will produce effects which are more or less serious.

#### THE THYROID GLAND

It is now recognized that the thyroid gland plays an important part in regulating the activities of the body. A decreased supply of its internal secretion, in the young, results in cretinism, with arrested development, indicating an interference with the normal nutrition of the body. In exophthalmic goiter, with an increased production of its internal secretion, there is an acceleration of tissue activity as shown by the rapid heart rate, loss of fat, anxious expression and general restlessness, indicating that nutrition is pushed beyond its normal rhythm.

The thyroid feeding experiments of Gudernatsch, upon tadpoles, showed that if thyroid, from any animal, was fed, there was a rapid metamorphosis into the frog stage. If fed in the very early stages frogs no larger than flies could be produced. Allen added the observation that if the thyroid be removed from a young tadpole it would not metamorphose into a frog, but would grow beyond the usual size of a tadpole and live a long time. If such a tadpole were fed with thyroid, it would promptly change into a frog. Under normal conditions it is believed that metamorphosis occurs when the nutritive conditions are ripe and the experiment would indicate that the thyroid had an influence, according to the quantity of its product, in either accelerating or retarding this ripening process.

#### THE PITUITARY GLAND

Dwarfism and gigantism (acromegaly) have, in recent years, come to be quite definitely associated with abnormal conditions in the pituitary gland. Like the thyroid the result depends upon whether there is a deficient or excessive amount of the secretion. The difference in size is evidently correlated with the processes of nutrition. There are other effects derived from pituitrin besides its influence on growth. It affects blood

pressure; it acts as a diuretic and also stimulates the flow of milk from the mammary gland. The quantity is not increased but the discharge of the milk that has accumulated is accelerated.

Some extraordinary results from the feeding of the fresh pituitary gland to poultry have been reported by Dr. L. N. Clark. Briefly, the results of two sets of experiments showed an increase in the laying capacity of approximately about 50%, with, in one experiment, an increase in the fertility of the eggs and the hatching of the chicks.

Sutherland Simpson carried out a similar set of experiments, following the methods of Dr. Clark as closely as possible, but with negative results so far as the laying capacity of the hens was concerned.

Emil Goetsch found that pituitary extract (anterior lobe), when fed to young rats, had a stimulating effect upon the growth of the animal and upon its sexual development and activity. The posterior-lobe extract, when thus given, had a retarding influence.

#### THE SEXUAL GLANDS

The sexual glands—ovary and testicle—possess both external and internal secretions. The former is concerned with the reproduction of the species; the latter regulates the male and female characteristics. The effect of castration, in either sex, not only changes the temperament of the animal, but leads in many cases to an accumulation of fat. The results will vary according to the age at which the operation is performed; but obviously the results are dependent upon changed conditions affecting the nutrition. Goetsch found that an ovarian extract (corpus luteum) had a stimulating influence upon the female, and a retarding influence upon the male, sexual development.

Although other examples might be cited, enough has been said to show that a most intimate relationship exists between the endocrine system and the normal processes of nutrition in the animal body.

Deficiency diseases are not confined to a lack of vitamins. There are also deficiency diseases resulting from an insufficient supply of the endocrine secretions, as well as other types of diseases due to an abnormally increased amount of the same secretions. Diseases due to an over-supply of vitamins have not yet been recorded. Although there are important differences there is this outstanding analogy, that both vitamins and certain

of the internal secretions (hormones) are essential for the nutritive processes concerned in the maintenance of life.

There is a growing belief, supported by some evidence, that each endocrine gland does not exist for itself alone, but that there is an interdependence and close relationship whereby the activity of certain of the glands is stimulated or inhibited by others.

*The therapeutic trail.* It has long been known that certain medicinal agents exert beneficial effects upon nutrition. Just how the action is brought about is not yet definitely understood. Independently of the alkaloids and other active principles that may exist in the plant, it is not at all unlikely that vitamins are also present and are soluble in the menstrua used in the preparation of fluid extracts, tinctures, etc. Although little or no work has as yet been done in this direction, it is by no means an unpromising field for research.

*The pathological trail.* In general, pathology is physiology gone wrong, complicated in certain instances by the addition of various infections. Elements of resistance or lack of resistance enter into the question and these elements are founded intrinsically upon the nutritive conditions of the animal. Pasteur showed years ago that the fowl which is normally not subject to anthrax could be made to acquire the disease by lowering its resistance by immersing one-third of its body in water cool enough to reduce its temperature a few degrees.

From what has previously been stated it may be readily inferred that pathological conditions may arise from disordered nutrition due to an insufficiency or lack of inorganic constituents, vitamins, or amino-acids in the food, or to an unbalanced condition of the endocrine system. Rickets, polyneuritis and scurvy are now quite definitely correlated with a deficiency of vitamins. Undoubtedly there are numerous other diseases correlated, in varying degrees, with the vitamin factor that have not, as yet, been definitely established. All of which emphasizes the necessity of considering the diet and its nutritional relations as contributing an important part to etiology.

#### PROFESSOR HART'S EXPERIMENTS

In the raising of stock the idea is quite commonly held that all of the requirements have been met when a balanced ration for the animal has been prepared, one which contains the proper proportion of digestible protein and energy-producing material. Experiments by E. B. Hart, of Wisconsin, demonstrate very clearly that

this idea needs modification. His experiments are most suggestive and have a wide-reaching importance in showing how a diet, supposedly correct, may disorganize nutrition sufficiently to disqualify the animal for any practical uses and, if the diet be continued long enough, probably to bring about a fatal termination. Professor Hart's work was really a test of the prevailing theory. Rations were balanced from the corn plant, wheat plant, oat plant and a mixture of the three.

In the first experiment some grade Shorthorns were purchased at the approximate weight of 300 pounds. They grew fairly well on all the rations, but it was noticed that those receiving the wheat were not so vigorous nor well developed as the others. If a corn-fed animal was changed to the wheat ration it became exceedingly stiff and if the wheat ration were continued long enough prostration resulted.

#### ALL-WHEAT RATION NOT SATISFACTORY

In another experiment upon some young, grade Holsteins, with the corn and wheat rations, it was observed that those fed on the corn ration gained in weight, grew well, matured, showed early estrum and were physically strong in all respects. All the evidence pointed toward normal nutrition. Those receiving the wheat ration grew only at a fair rate and, when they reached a weight of 1000 lbs., not only ceased to grow but lost weight and showed no vigor. There was physical weakness and blindness ultimately occurred. On coming into their stalls, these animals would collapse under the slightest excitement, show labored respiration and more or less trembling. After a few minutes the animals would be able to rise. They exhibited no estrum and were obviously of no use for breeding purposes. Emaciation and loss of weight became so marked that it was deemed necessary to kill them.

When the Shorthorns of the first experiment were old enough for reproductive purposes, it was found that the animals on the corn ration carried their calves to maturity, had easy parturitions and produced strong and vigorous calves. Those fed on the wheat ration did not reproduce normally. The calves were born twenty-five or thirty days before term. They were undersized, weak and never lived more than four or five days. The dams frequently failed to clean properly and were exposed to the danger of infection. Some of them died from this cause.

The animals on the oat ration produced fairly good offspring,

but in no case did they appear quite so vigorous as the offspring from the corn-fed mothers.

Throughout the experiments the herd was under the observation of a veterinarian and no evidence was found of the contagious form of abortion disease.

Professor Hart found that if corn grain were fed along with wheat straw the offspring were weak or dead at birth, but if to this diet a suitable salt mixture was added then perfect offspring resulted. Obviously one of the difficulties with the wheat diet was an insufficiency of the mineral constituent.

If the same suitable salt mixture was added to the wheat ration consisting of wheat grain and wheat straw, then disaster again ensued. Evidently something more than the salts was required and this something Professor Hart believes to be a toxic substance present in the embryo of the seed.

#### PROFESSOR HART'S CONCLUSIONS

From the results obtained it would appear that the following conclusions are worthy of consideration:

1. That weak or dead offspring may result from nutritional disturbances caused by the continued use of certain natural food substances in a balanced ration.
2. That rations concerned in producing premature birth are likewise concerned in the failure of the animal to "clean" properly, with the attending danger of infection.
3. That an excess of material like wheat straw in the ration is an important factor in causing premature birth because of its low salt content and the possible presence of a toxic substance.

As an example of susceptibility to other diseases, because of lowered resistance due to the nutritional disturbance, it was reported that in an outbreak of anthrax in the University herd, the only loss that occurred in the experiment group was among the wheat-fed animals.

In these particular experiments it is difficult to avoid the conclusion that the abortion was a deficiency trouble. If the mother was in an unsatisfactory nutritive condition, it could hardly be expected that the task of developing a new structure, from its own inadequate supply of nourishment, would result successfully.

The existence of a vitamin factor concerned with reproduction is not beyond the realm of possibility, and this question is receiving the attention of scientific workers. Nutrition must be considered in connection with abortion. Even in the infectious type,

the diet should, by all means, receive attention as to its influence in lowering or increasing resistance to the disorder.

There is no other period in life when the growth factor is so pronounced as the few weeks or months following birth. During the suckling period, if the mother is in a state of health, it may be assumed that the milk represents a perfect food for the nutritional requirements of the young. All mammals are lactivora up to the weaning period. The transition from milk to another kind of food during the process of weaning affords many opportunities for disordered nutrition. The growth factor, nevertheless, seems to be so strong that even when the diet is unsatisfactory some progress may be made.

Professor Hart's experiments brought out the important point that in the wheat-fed group, in spite of its handicaps, the animals gained 700 lbs. before disaster occurred. The important inference also seems to be justified that an unsatisfactory ration may be continued for a relatively long time before its deleterious effects become marked and, because some nutritional progress is made, the effects may be attributed to other or unknown agencies. The animal organism is adaptable and will submit to abuse for varying periods according to the strength of the stimuli. In the case of weak stimuli a cumulative action over a long period may be necessary until sufficient momentum is acquired to bring about a reaction. It is of the highest importance that we should know the diet and its effect upon nutrition.

#### DIABETES A DEFICIENCY DISEASE

One of the oldest nutritional diseases is diabetes, occasionally encountered in the dog, but only too commonly found in the human race. Until recent years it has defied all scientific effort to subjugate it. It is now known that the disease is associated with a deficiency of the internal secretion of the pancreas. In this sense it is a deficiency disease. It is now believed that the abnormal condition or lack of this secretion interferes with the glycogenic function of the liver and likewise the muscle and other tissues, by inhibiting their power of utilizing the food sugar brought to them by the circulation. The recent discovery of insulin and its application to this disease presents some points of analogy to the thyroid deficiency in cases of cretinism. By supplying the missing material improvement results, which, unfortunately, is not permanent; for, on discontinuing the treatment, the symptoms reappear.

Since, in this disease, the tissues are unable to consume carbohydrates, the fat is called upon to supply what nourishment it can. In the normal body the fat undergoes complete combustion to form carbon dioxide and water. It is necessary that some carbohydrate should be burned with the fat in order to obtain this result. In diabetes the carbohydrate is not available in the tissues for this purpose and fat burns incompletely with the formation of certain fatty acids which aggravate the disturbed nutrition still more by causing acidosis. In incomplete combustion the comparison has been made to a fire burning with considerable smoke because of the absence of the carbohydrate fuel—the smoke being represented by the abnormal fatty acids. In all natural fats it appears that the carbon atoms of the fatty acids are arranged in even numbers and in the process of combustion these atoms are split off in pairs until a 4-carbon-atom stage is reached—butyric acid. It is from this point that the smoke begins—the production of the fatty acids which cause acidosis.

#### EXPERIMENTS WITH INTARVIN

A synthetic fat, with an odd number of carbon atoms, has recently been introduced by Dr. Max Kahn, under the name of intarvin, for the treatment of diabetes and the prevention of acidosis. It is said that beneficial results have been obtained in a number of cases. The principle involved is to prevent the 4-carbon-atom stage (butyric acid). If the carbon atoms are split off in pairs, there will be no butyric acid and its products which cause acidosis, because there is no stage in which the carbon atoms exist in an even number.

Experiments in our laboratory show that intarvin is digestible and that its ingestion is followed by an increased number of chylomicrons in the blood.

It has been said that there is a high percentage of sickness in calves, especially among the pure-breds, during the nursing period and up to the age of two or three months. Commonly the trouble is in the form of digestive disorders, ranging from scours to mild diarrhea and associated with low resistance, during which various infections may gain lodgement and cause more or less serious results.

In an effort to get back to nature, it is well to consider the ancestral cow. Her milk, like that of other mammals, was intended only for the nourishment of her offspring. It is fair to assume that if an analysis of the milk of the ancestral cow were

available it would show some surprising differences when compared with that of the modern dairy cow. By a process of selection, attention to rations and various artificial conditions, it has been possible not only to increase the milk production enormously but along with this a certain amount of increase of the different constituents, particularly the butter fat. Is the digestive apparatus of the modern calf essentially different from that of the ancestral calf? Has Nature endowed the modern calf with a more powerful series of digestive enzymes to cope with the richer and more abundant supply of its diet? We do not know. We do not even know if all the enzymes which are in evidence later are present at the time of birth. In the human infant it is stated that the amylolytic enzyme of the pancreas does not appear until the first month of life.

If the modern calf, with the digestive apparatus of an ancestral calf, has to contend with a diet radically different from the ancestral diet, then it is not difficult to understand why the modern calf is subject to digestive troubles and disordered nutrition. In sheep and swine and other animals, not bred for milk production, troubles of this kind are rarely encountered.

The transition from a milk diet to solid food, although common to all mammals, is more or less of a critical period in taxing the digestive enzymes, some of which may have been dormant or absent earlier, with unaccustomed work and must necessarily influence the nutritive processes. In the herbivorous animals this diet metamorphosis is accompanied by a change in the urine from an acid to an alkaline reaction with certain changes in the urinary constituents. As a result of metabolism various acids are normally produced in the body and the urine is one of the channels of escape for some of the acid products. A diminished flow or suppression of the urine, by retarding or preventing their elimination, would favor the accumulation of the products in the body and thus serve as a factor in causing acidosis.

#### ACIDOSIS AND DIABETES

Although acidosis was first studied in connection with diabetes and its relation to the disordered nutrition characteristic of that disease was clearly established, later study has shown that acidosis may occur independently of diabetes and that it may occur in varying degree and vary as to its cause, although fundamentally concerned with nutrition. It is not the mere presence of acetone bodies, as in diabetes, that determines acidosis. In

the young they may not be a frequent cause.

The reaction of the blood is slightly alkaline. The constancy with which this reaction is maintained is most extraordinary. It is desirable to keep in mind the extreme sensitiveness of the enzyme and chemical actions in the body, which are interfered with or absolutely inhibited by very minute changes of reaction in the various fluids. A change in the reaction of the blood from normal to precise neutrality is sufficient to render life impossible. A change from the reaction of ordinary drinking water, which is more alkaline than the blood, to that of distilled water, which is much more acid than blood, would be incompatible with life.

The important constituents of the blood, so far as the regulation of its reaction is concerned, are: sodium bicarbonate, occurring both in the plasma and in the cells; the acid and alkaline phosphates of sodium and potassium, found almost entirely in the red blood cells, and the proteins. These three measures of defense act in cooperation with each other and are present in the blood itself. There are probably other means of defense in the tissue juices and cells of the body. There is a further means of defense in that the body is able to neutralize acid by the production of ammonia. Normally, a small amount of this alkali is always formed, but if acids are introduced into, or formed in, the body in unusual amount, the danger is met by an increased production of ammonia.

In the child acidosis is often associated with recurrent vomiting and diarrhea. In the calf, true vomiting is difficult or impossible but diarrhea is not uncommon and is doubtless associated with a disturbed metabolism. The defensive mechanism becomes unbalanced with resulting acidosis. In such cases there is usually an increased alkali tolerance and beneficial results have been obtained by the administration of sodium bicarbonate in an amount sufficient to render the urine alkaline.

#### CALF MORTALITY HIGH

The percentage of sickness and mortality among calves is relatively high and some of the causes may antedate the birth of the calf itself, as suggested by the experiments of Professor Hart. Other causes may relate to the digestive capacity of the calf and its possible inadequacy to cope with the modern milk standard, and the transition from the milk to solid food before the suitable digestive enzymes are present or have developed sufficiently to digest the new material properly. All of these trails lead to

nutrition which, when once unbalanced, paves the way to lowered resistance and opens the door to various infections.

Much of what has been said has been merely suggestive. Some of the trails are tangled and unexplored but it is possible to follow them to some extent and, at least partially, to clear them.

In the old days all roads led to Rome. In physiology, and much of pathology, all trails lead to nutrition. Let us not forget that back of nutrition is light. At our dining table we may consume Florida sunshine in our grapefruit; California sunshine in our lemons and oranges; Dakota sunshine in the wheat we eat, and possibly South American sunshine in beef imported from Argentina.

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#### "PINGY" PROCURES PUBLICITY

Paderewski's pedigreed pet Pekingese "Pingy" has surely been getting publicity. A Peoria paper published a press dispatch proclaiming, "Paderewski's Pingy Preferred to Piano." The Chicago papers, of course, printed profuse articles about "Pingy," suggesting that he probably had a proficient press-agent pounding out propaganda while the palatial private car of the prominent Polish pianist was parked in Packingtown. The pampered "Pingy" caught cold while promenading on the prairies of South Dakota. The post-war Premier of Poland piked back to Chicago to get professional care for "Pingy." Dr. W. P. Tague was selected as chief of staff, with a plentiful supply of consultants. A "flock of nurses paraded on board," according to one press dispatch. Another stated that "Pingy" had been pronounced dead at 4 o'clock in the afternoon, followed by another, that same evening, to the effect that he was putting up a fight for life. The papers even published the prescriptions of the pink pills, potions and powders prescribed for the ten-pound pooch—Ping Lung, to name him properly. He is said to have been the precious property of a son of the last emperor of China.

## DIVERTICULITIS IN SWINE

By L. P. DOYLE

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Diverticula of the colonic mucous membrane constitute a conspicuous histologic feature of the normal colon of swine (fig. 1). Pathologic changes in these diverticula give rise to prominent gross lesions that are especially discernible when the abdominal cavity is first opened and the serous surface of the colon exposed to view. The true nature of these lesions appears not to have been

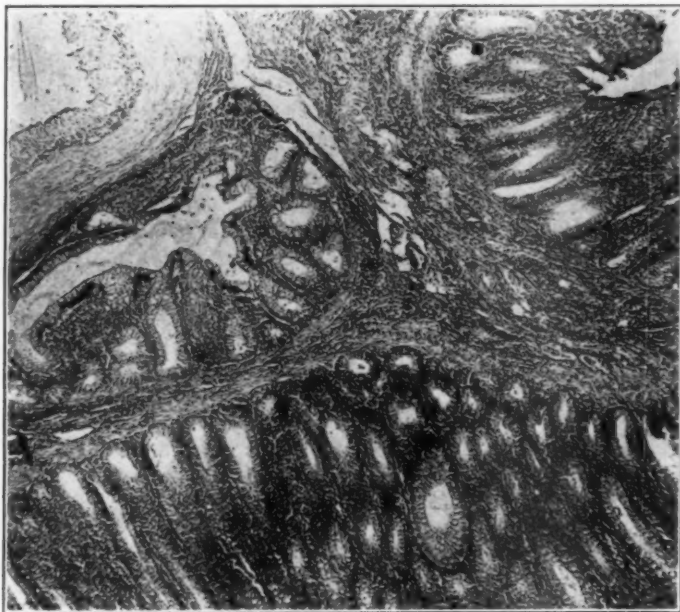


FIG. 1—Photomicrograph of a section of a hog's colon showing a normal diverticulum in the submucosa.

recognized. They apparently have been misinterpreted as indicating invasion of the colon wall by verminous parasites. The microscopic examination of sections of a large number of colons showing these lesions has shown that the essential changes occur in the diverticula, and that metazoan parasites do not bear any demonstrable relationship to the lesions commonly found.

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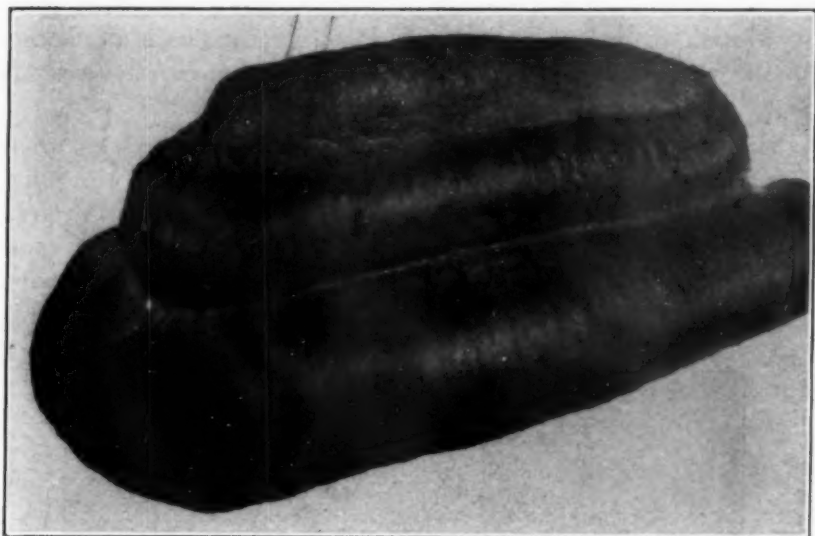


FIG. 2—The gross appearance of a hog's colon showing diverticulitis. In this stage, the diverticula contain caseous material

The macroscopic appearance of the lesions depends upon the stage of the pathologic change in the diverticula. What appears to be the earliest recognizable stage in the formation of the gross lesions is indicated by circular, clear, hyaline areas resembling grains of cooked tapioca in the colon wall. A later stage in the

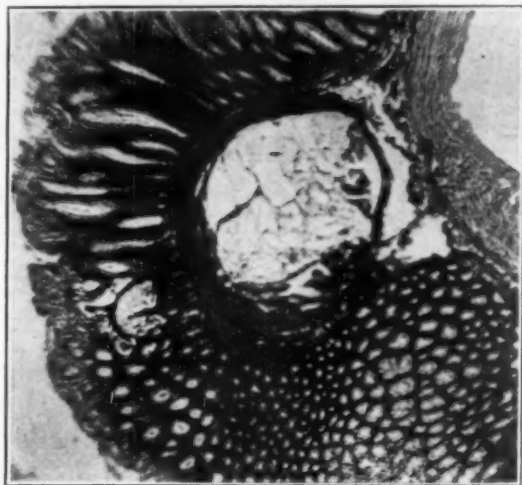


FIG. 3—Photomicrograph of a diverticulum in the mucous stage of degeneration. Outlines of the crypts may still be seen in the diverticulum. In this stage of degeneration the diverticulum has the gross appearance of a circular, clear, hyaline area, resembling a grain of cooked tapioca, in the colon wall.

pathologic process is indicated by the presence of spherical, gray or whitish, caseous masses, about 2 or 3 millimeters in diameter, in the wall of the colon (fig. 2). These lesions in the caseous stage have been referred to as "spherical abscesses."

Histologic examination of a lesion in an early stage, i. e., when the gross appearance is that of a clear, hyaline area in the colon wall, shows mucous degeneration of the epithelium and a large amount of mucus distending the diverticulum (fig. 3). Examination of a later stage shows the diverticulum to be filled with closely packed leucocytes and cellular débris (fig. 4). In this



FIG. 4.—Photomicrograph of a section of a hog's colon showing a diverticulum filled with leucocytes and cellular débris. The communication between the diverticulum and the lumen of the colon is also plainly shown. In this stage, the diverticulum has the gross appearance of a "spherical abscess," filled with caseous material.

stage there may not be any epithelium found in the diverticulum except a lining made up of squamous cells.

These pathologic changes in the diverticula frequently occur as a part of a general colitis. However, they have been observed to occur where there was not any recognizably pathologic change in the mucous membrane along the lumen of the colon.

Diverticulitis has been observed in dysentery in swine, in hog cholera, and in entero-colitis resulting from the feeding of a pure culture of *B. suispestifer*. It occurs quite constantly in the

condition, or group of conditions, commonly known as "pig typhoid."

#### CONCLUSION

The circular lesions that are found so frequently in the colon wall of hogs represent pathologic changes in diverticula of the mucous membrane. They are not evidence of esophagostomiasis or other forms of parasitism. No specific diagnostic significance can be attached to these lesions alone, because they may occur in any one of several diseases that are etiologically distinct.

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#### VETERINARY EVOLUTION

Long years ago, as we all know,  
The Vet. was quite a quack.  
His fav'rite ills, which called for pills  
And caused his brain to rack,  
Were "hollow horn," for which he'd sworn  
Red pepper was specific.  
"Wolf-in-the-tail," he'd never fail  
To cure—with pain terrific.  
With "dry murrain" he's often slain  
Great herds of burly bovines.  
With methods cruel, in his hard school,  
He studied holding side-lines.

#### II

But the present day, we're forced to say,  
Has made the Vet. anew.  
He knows his stuff quite well enough,  
And knows just what to do.  
With all his might he makes his fight  
To cure a poor dumb pet.  
In ways humane he eases pain,  
So don't forget the Vet.  
When Dobbin's sick, he'll do the trick,  
And coax his health right back.  
When stock gets thin, just call him in,  
But don't call in a quack.

—*Knight Awdlee Hughes.*

## ASCITES IN THE DOMESTIC FOWL<sup>1</sup>

By B. F. KAUPP and R. S. DEARSTYNE, *Raleigh, N. C.*

*From the Laboratory of Poultry Pathology, North Carolina Experiment Station and Agricultural College*

### INTRODUCTION

Ascites is a collection of serous fluid in the abdominal cavity. Ascites proper is dropsy of the peritoneum characterized by increased size of the abdomen, fluctuation, and general signs of dropsy. It is rarely a primary disease, usually owing to obstructed circulation in some of the viscera or excitement of vessels of the abdominal organs.

Ascites may accompany cirrhosis of the liver, tubercular peritonitis, general chronic peritonitis, organic heart disease, or chronic renal troubles.

According to their etiology there are five varieties of edemas, as follows: edema due to arterial congestion; edema from stagnation of the blood; edema caused by a hindrance to the outflow of the lymph; edema caused by a disturbance of the capillary secretions due to changes in capillary walls; and edema ex-vacuo.

### WHAT AUTOPSY RECORDS SHOW

In a review of 800 autopsies from the records of this laboratory 20 (2.5%) were suffering from abdominal dropsy. The amount of fluid present in the abdominal cavity at the time of autopsy varied from 20 cc to 1100 cc. The following is a tabulation of the amount of ascitic fluid in each case.

TABLE I

Case No.	Amount of Fluid	Case No.	Amount of Fluid
1		2	400 cc
3	250 cc	4	50
5	50	6	250
7	20	8	....
9	100	10	260
11	....	12	....
13	600	14	225
15	430	16	250
17	75	18	25
19	1100	20	30

The body weight of no. 19, exclusive of the ascitic fluid, was 1735 grams or 62% of the total weight of the bird.

The following is an account of one case of ascites in a hen:

Received for publication, February 25, 1924.

*Subject:* Barred Plymouth Rock hen.

*Source:* Brought to hospital by Mrs. A., of Raleigh, N. C.

*Clinical Studies:* Patient was brought to the hospital April 18, 1923. Excessive appetite, normal weight, happy disposition with bright plumage. The abdomen was enlarged, rather pendulous and tense as shown in the accompanying photograph (fig. 1).

On entering the hospital there was drawn from the abdominal cavity, 600 cc of turbid ascitic fluid. On May 10, 22 days later,

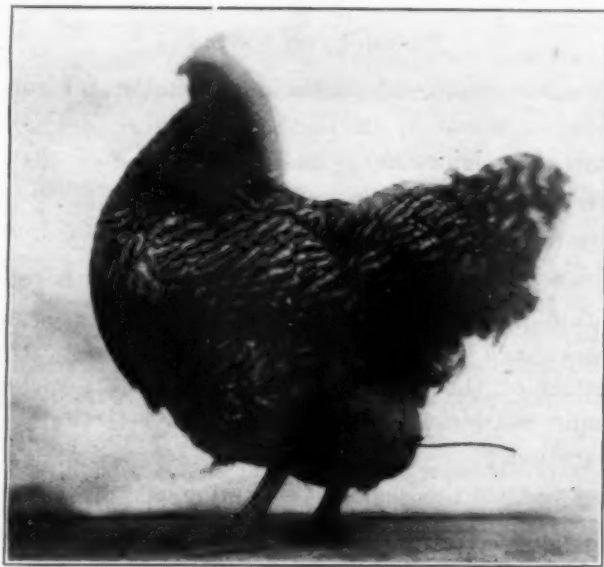


FIG. I. A Barred Plymouth Rock hen with ascites.

there was drawn from the abdominal cavity, 300 cc of ascitic fluid. On June 21, 42 days later, there was drawn from the abdominal cavity, 300 cc of ascitic fluid, or 13.6 cc per day. In the second period of 42 days there was poured out into the abdominal cavity, 460 cc, or 10.9 cc per day.

TABLE II  
RESULTS OF BLOOD STUDY OF HEN WITH ASCITES

Numerical Count			Differential Count				
Date	Leucocytes	Erythrocytes	Poly.	Lymphocytes		Mast	Hemo- globin
				Small	Large		
6- 7-23	24,000	3,670,000	31	29	40	..	95
6-19-23	33,000	3,090,000	33	29	34	4	98
6-20-23	37,000	3,350,000	34	23	41	2	95
6-21-23	30,000	3,340,000	32	32	36	..	95

It is quite apparent that more fluid finds its way into the abdominal cavity for the first few days after drainage of the fluid than later, when the accumulated fluid is sufficient to cause considerable pressure against the surrounding structures.

The blood coagulates in one minute, showing that the time for coagulation had been doubled. The normal time of coagulation of the blood of an adult hen, in health, as we have determined in this laboratory, is one-half minute. All cultures of ascitic fluid proved negative.

#### POSTMORTEM FINDINGS

The hen was destroyed, in the gas chamber, for autopsy purposes.

The carcass weighs 2350 grams.

Culture tests of blood are negative.

Culture tests of ascitic fluid are negative.

The liver weighs 75 grams, with possible slight hepatitis.

The kidneys, heart, spleen, and intestines appear normal. The fibers have not lost their cross striation. Microscopic examination of the kidneys and spleen was not made. On microscopic examination the liver showed fatty degeneration and congestion.

Culture tests of the heart, liver, and spleen are negative.

There is present in the intestine one *Ascaris inflexa* with heavy infestation with *Taenia infundibuliformis*.

#### SUMMARY

There is here given a record of hospital and autopsy study of a case of ascites in a Barred Plymouth Rock hen.

The blood study is that of a normal fowl. The blood is tardy in its power to coagulate.

There accumulates, in the abdominal cavity, ascitic fluid at the rate of 10 cc to 13 cc a day.

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#### AN ERROR CORRECTED

In the list of members of state veterinary examining boards, published in the April issue, Dr. F. F. Meads should have been listed as secretary of the Oklahoma Board, instead of his brother, Dr. E. W. Meads.

## BACILLARY WHITE DIARRHEA IN POULTRY<sup>1</sup>

By H. R. BAKER, Newark, Del.

Dept. Animal Industry, University of Delaware.

In 1900, Rettger reported his studies on a disease of young chicks, highly common in this country and sometimes exceedingly destructive, manifested by whitish diarrheal discharges (whence the name, "white diarrhea") and characterized by a generalized bacteremia. Rettger isolated from cases of this disease a bacillus which he and his collaborators have amply demonstrated as the cause of the most common form of the malady, and which Rettger called *Bact. pullorum*, because it was found as the cause of disease in young chickens. Since 1900 Rettger's observations and conclusions have received wide support and confirmation on the part of many bacteriologists in this country. Our knowledge of the nature of the disease, its manner of transmission and its causative agent has been much advanced. During that same year, Rettger succeeded in producing the disease in young chicks by subcutaneous injections and by feeding the cultures. The organism was recovered from those that died of the disease.

Rettger and Stoneburn (1909) found *Bact. pullorum* in the eggs and pathological ovaries of adult fowls.

Jones (1909) also carried out a number of experiments. In his inoculation experiments chicks twenty-four hours old were fed pure cultures of *Bact. pullorum*. The average mortality amounted to 82.5%, while in the controls the mortality amounted to but 2%. In this report it was also stated that infection may take place in three ways, that is: (1) Through the egg; (2) by direct contact, in the incubator, with chicks that have acquired the disease through egg infection; (3) by chicks being placed in contaminated surroundings. He also stated that chicks are most susceptible to infection during the first twenty-four hours of life.

Gage (1911) examined a number of adult hens dying of unknown causes and found *Bact. pullorum* in the ovaries. He states that some of the ova were gangrenous.

Jones (1911) clearly proved that a number of adult females that had been infected when young chicks and had overcome the

<sup>1</sup>Presented at the annual meeting of the Delaware Veterinary Medical Association and University Veterinary Conference, Newark, Delaware, Dec. 19th, 1923.

disease became harborers of the organism. *Bact. pullorum* was found in the eggs and ovaries of some of them. The ovaries of those harboring the organism were found to be pathological. In this report it was also shown that the local disease in the ovary of adult hens could be produced by the intravenous injections of 1.5 cc of a broth culture of *Bact. pullorum*. Jones further suggested the use of an agglutination test as a method for detecting fowls that are harboring *Bact. pullorum* in the ovary.

In 1912, Jones observed an outbreak of an acute disease in a flock of adult fowls. This outbreak lasted about one month and, in all, fifty fowls died of the disease. Cultures from the liver, spleen, heart, and ovaries revealed the presence of *Bact. pullorum* in pure cultures. The disease was a true septicemia having a somewhat constant group of lesions. These lesions usually were minute necrotic foci in the liver, spleen, and pancreas, and larger necrotic nodules in the heart muscle. Jones concluded that, under certain conditions, *Bact. pullorum* seems to be pathogenic for adult hens. At this same time, Jones examined the ovaries of 21 fowls that reacted to the agglutination test. *Bact. pullorum* was isolated from each of the ovaries, but only 15 of the 21 ovaries were found to be cystic. These findings indicate that the ovaries of fowls harboring *Bact. pullorum* are not always pathological. An examination of all hens which were non-reactors revealed normal and bacteriologically sterile ovaries.

Beaudette and Bushnell, of the Kansas Station, have carried on experiments during the past two years on the relation of *Bact. pullorum* to hatchability and fertility of eggs. The agglutination test applied to a flock in which poor hatching was reported showed that 10.4% of the fowls were infected with *Bact. pullorum*. It was shown that the fertile eggs from the infected hens of this flock gave a 45.2% hatch, whereas the fertile eggs from non-infected hens of this flock gave a 63.2% hatch. The fertility of the eggs from the infected hens was 57% as compared to 90.4% fertility for non-infected hens. These results show that eggs from infected hens may be infertile; may produce a dead embryo, usually at about the 19th day; or may produce a chick which lives but suffers from white diarrhea.

Chickens hatched from eggs which are free from the white diarrhea organism have a better chance to live. Gage, of Massachusetts, obtained records of the viability of chickens from white-diarrhea-free flock "A" furnished to seven different poultry

owners. All these seven poultrymen said their losses usually were about 50%. The percent of viability with the white-diarrhea-free chicks was raised to 92%, the individual flocks varying from 80% to 98.5%.

Massachusetts, along with a few other states, has been carrying out a campaign for the eradication of white diarrhea for several years. This station reported in 1915 that no chickens died of white diarrhea in a flock of 1000 chickens hatched from eggs from non-infected hens. During the previous season, before the bearers of infection had been eliminated from the flock, only 200 chicks of 2000 hatched survived the ravages of the disease.

In 1916, the Massachusetts Station tested ten flocks in different parts of the state. The percentage of reactors varied in the various flocks from 6.6% to 50.0%.

From 1915 to 1918, 26,000 birds were tested in Massachusetts. Thirteen and one-half percent reacted positively to the test. During 1920, 25,000 birds were tested with 12.5% reactors. In 1921, 30,000 were tested, with 12.6% reactors. In 1922, 33,000 were tested with 7.6% reactors.

The results obtained in this laboratory for Delaware flocks for last year are given in table No. 1.

TABLE NO. 1—RESULTS OBTAINED IN DELAWARE  
1922

Flock No.	No. Tested	No. Reactors	% Reactors
1	537	19	3.5
2	167	15	8.9
3	200	58	29.0
4	130	3	2.3
Total	1034	95	Aver. 9.2

1923

5	115	4	3.5
6	248	27	10.9
1	261	0	0
7	218	29	13.1
4	87	0	0
8	54	6	11.1
3	131	10	7.6
9	126	26	20.7
Total	1240	102	Aver. 8.2

It is interesting to note the reduction in the number of reactors in the flocks which were tested both years. Flock no. 1 reacted 3.5% in 1922; this year there were no reactors. Flock no. 3 reacted 29% last year; this year there were 7.6% reactors. Flock no. 4 reacted 2.3% last year; this year there were no

reactors. These last results confirm the findings of Gage in Massachusetts. Gage found that it took two or three years to eliminate all reactors from a highly infected flock. However, Gage reports that when new stock is introduced into the flock there is a grave danger of renewing the infection. He cites an example of a flock under his observation. The first year the flock was tested, 10% reacted. This was reduced to 2% the second year. The third year the flock was not tested and new stock was introduced in the form of show birds bought from a prize-winning strain. The fourth year the flock was again tested, and the owner was surprised to learn that 14.5% of the birds reacted.

To summarize briefly the dangers of the presence of *Bact. pullorum* carriers in the flock and the advantages in eliminating these carriers from the flock, the following facts should be brought out. First, the mortality of chickens hatched from eggs of *Bact. pullorum* hens is very high. Second, the fertility of the eggs is lower and death of the embryo is higher in eggs from infected flocks. Third, the hen with an infected ovary is not such a high egg-producer as a normal fowl. Fourth, there is always the danger of the organism becoming pathogenic for the hen itself and causing an infectious outbreak of the disease in the whole flock. Fifth, the carrier of *Bact. pullorum* is usually eliminating the living germs from her body and spreading the infection to healthy chickens.

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### BUREAU TRANSFERS

Dr. C. W. Chapin (Chi. '15), formerly stationed at Baton Rouge, La., is now at Columbia, S. C., with the B. A. I.

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Dr. Roy F. Gard (K. C. V. C. '10) has been transferred to Topeka, Kans., from Kansas City, on virus-serum control.

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Dr. Ernie C. Hughes (Ind. '16) has been transferred from Lincoln, Neb., to Chicago, on tuberculosis eradication work.

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Dr. Joseph B. Clancey (Chi. '92) has been transferred from Jacksonville, Ill., to Omaha, Neb., on meat inspection work.

---

Dr. Jefferson Robinson (K. C. V. C. '15) has been transferred from Kansas City to Jacksonville, Ill., on meat inspection work.

---

Dr. L. J. Allen (Ont. '95) has been transferred from Oklahoma City to Ft. Worth, Texas, on tuberculosis eradication work.

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Dr. Harry Grafke (K. C. V. C. '07) has been transferred from Fort Worth to Oklahoma City, on tuberculosis eradication work.

## SOME VETERINARY REMINISCENCES

### I. Testing the Tick Theory

*By N. S. MAYO, Chicago, Ill.*

It has been said that when one grows reminiscent it is a sign of advancing years or failing mentality, yet in the face of this I am going to tell some of my experiences as a veterinarian in the Central West some thirty-odd years ago. They are not unusual experiences, everyone has had them, but few have written them down. Maybe these experiences will prove interesting and suggestively helpful to some young practitioners now, or they may stimulate others to write of more important episodes of days that seem "far away and long ago."

Having completed a course in the Michigan Agricultural College, armed with a bachelor's degree, Prince Albert coat, plug hat and black-and-white checked "pants," I blew into the Chicago Veterinary College. I have marvelled ever since that the gang that was supposed to study veterinary medicine there did not string me up to the nearest telegraph pole on State Street. I have reformed and have never been arrayed in such raiment since.

At the time I graduated in veterinary medicine the profession was thoroughly stirred by a bitter controversy between Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, and Dr. F. S. Billings, over the causative agent of hog cholera. Dr. Billings, a brilliant but erratic student, had been educated in Germany. He had been on the staff of the Nebraska Experiment Station, but had been compelled to resign because of his eccentricities.

There was a striking change taking place in veterinary lines. The great live stock industry of the West was beginning to recognize the importance of protecting their flocks and herds against transmissible diseases. The Bureau of Animal Industry had been recently established and with the assistance of state authorities had stamped out contagious pleuro-pneumonia. Smith and Kilbourne were doing their classic work on Texas fever and tuberculosis was beginning to be considered as a serious problem to the cattle industry.

Experiment stations were being organized under the recently enacted Hatch law and there was a demand by the colleges and experiment stations in the West for veterinarians to do research

work on animal diseases. As a result of this I was in 1890 appointed professor of veterinary science in the Kansas Agricultural College and veterinarian to the Experiment Station there. Dr. Francis had recently gone to the Texas College, Dinwiddie to the Arkansas Station, Paul Paquin to the University of Missouri, and the live stock men soon compelled the University of Nebraska to reinstate Dr. Billings. As I recall, Dr. A. A. Holcomb was state veterinarian of Wyoming and I presume Dr. Lamb held the same position in Colorado. I've never found an old inhabitant who could recall any other state veterinarian in Colorado. A Dr. Going, M. R. C. V. S., was state veterinarian of Kansas.

About 1890 the "tick theory" had been announced, but there were a good many "doubting Thomases" in Missouri and adjoining states. Dr. Francis, of Texas, suggested that we try the theory out. He was in a tick-infested region and Kansas was free, so he sent me some tick eggs in a bottle by mail. I hatched them out and placed some of the young ticks on a couple of calves without any serious results. I expressed my disbelief in ticks to Dr. Francis and he suggested that I put the young ticks on an older animal. He sent me another batch of eggs. I was so skeptical that I allowed about forty young ticks to crawl onto my own family Jersey cow. When I hauled her out and performed the last sad autopsical rites I felt that there must be something in the tick theory.

### BUREAU TRANSFERS

Dr. C. S. Parks (Ind. '21) has been transferred from Albermarle to Wadesboro, N. C.

Dr. Wm. R. Scott (K. C. V. C. '12) has been transferred from Chicago to St. Louis, on meat inspection work.

Dr. E. P. Johnson (McK. '06), stationed at Sacramento, Calif., has been transferred to Albuquerque, N. Mex.

Dr. Geo. J. Mutziger (K. C. V. C. '05) has been ordered to South St. Joseph, Mo., from St. Louis, on B. A. I. work.

Dr. Robert N. Ashley (K. C. V. C. '08) has been transferred from South St. Paul to Albuquerque, New Mexico.

Dr. Edward C. Jespersen (McK. '16) is now located at Sioux City, Iowa, on meat inspection work for the B. A. I.

Dr. L. M. Buffington (Cinn. '11) has been transferred from Ames, Iowa, to Des Moines, on hog cholera control work.

## A NEW NEMATODE, *PROTOSPIRURA GRACILIS*, FROM THE CAT<sup>1</sup>

By ELOISE B. CRAM, Washington, D. C.

Zoological Division, U. S. Bureau of Animal Industry

Some nematode larvae that had been collected from a dung beetle, *Aphodius fimetarius*, were received alive in the laboratory of the Zoological Division of the U. S. Bureau of Animal Industry, from the laboratory of Dr. N. A. Cobb, U. S. Bureau of Plant Industry. These larvae were fed to a cat and two months later the cat was chloroformed and examined postmortem. In the stomach a single nematode was found, an adult male, a description of which follows:

*Protospirura gracilis*, new species.

**Male:** Slender worm, 23.2 mm. in length with a greatest width of  $564\mu$ . Diameter of head  $116\mu$ . The mouth has two large lips, each divided into three lobes, the median ( $41.5\mu$  wide) being larger than the laterals ( $30.7\mu$  wide). Each lobe has on its inner surface several teeth; it was difficult with the limited material to determine the number. The median lobe appears to have seven teeth, of which the one in the median line and the two projecting at the margins are larger than the two pairs situated between the median and these laterals (fig. 2). Each lateral lobe of the lips appears to have three

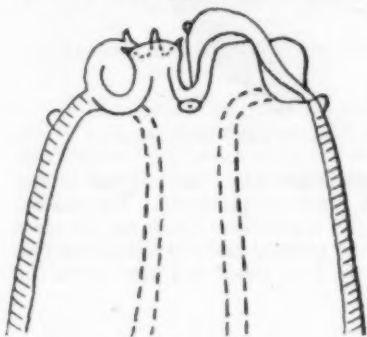


Fig. 1. *Protospirura gracilis*. Anterior extremity, oblique view.

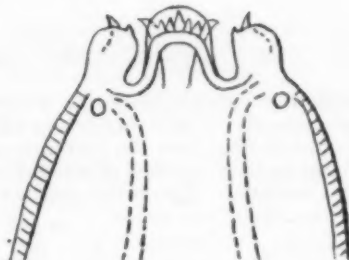


Fig. 2. *Protospirura gracilis*. Anterior extremity, lateral view.

teeth, a large median and two smaller laterals, all three projecting beyond the margin of the lobe (fig. 1). There are four cephalic papillae, one at the base of each lateral lobe near its junction with the median (figs. 1 and 2). The pharynx, with heavily chitinated walls  $8\mu$  thick, is  $141\mu$  deep; the diameter of its lumen increases from  $42\mu$  near the mouth to  $51\mu$  near the base. The esophagus is 2.3 mm. long; its maximum thickness is  $232\mu$ . The nerve ring

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is situated  $365\mu$  from the anterior end of the esophagus. The posterior extremity (fig. 3) is tightly coiled in a spiral. The ventral surface exhibits the oblong cuticular elevations arranged in longitudinal rows, markings which are similar to those of other species of the genus. There are two bursal alae of equal length ( $996\mu$ ); their maximum width of  $66\mu$  is attained just anterior of the middle. They bear coarse, wavy, transverse striations, which produce a fringed appearance at the margins. The spicules are of unequal length; they are narrow but their proximal extremities are somewhat dilated, that of

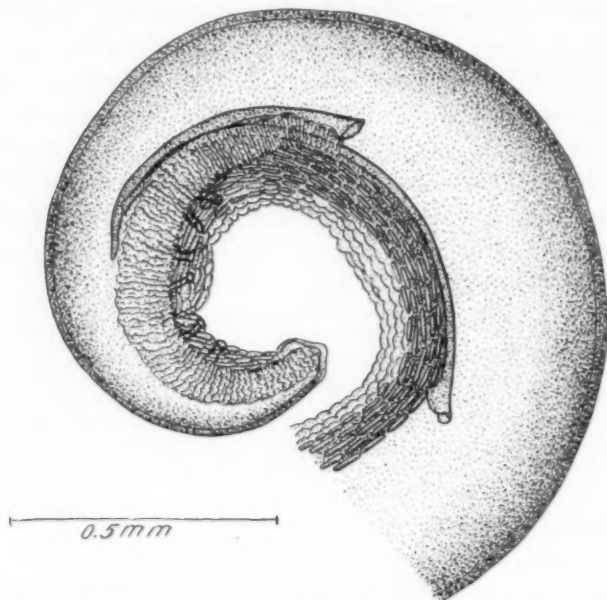


Fig. 3. *Protospirura gracilis*. Posterior extremity of male, lateral view.

the shorter spicule presenting a ragged appearance while that of the longer one is smooth. Their lengths are  $622\mu$  and  $1.1$  mm. respectively. The cloacal aperture is  $332\mu$  from the posterior extremity of the body. There are six pairs of large stalked papillae, of which four pairs are preanal and the remaining two pairs postanal. The testis extends anteriorly to a point  $5.5$  mm. from the anterior end of the worm.

Female: Unknown.

Host: *Felis domestica*.

Location: Stomach.

Locality: Washington, D. C.

Just previous to being chloroformed, the cat in which the worm was found had eaten part of a white rat, (*Mus norvegicus albus*), and this was still undigested in the stomach. It is questionable whether the cat was the true host of the worm, or whether it had merely ingested a rat parasite with the rat that it had eaten. However, in postmortem examinations of a series of 113 white rats, reared under the same conditions as the one in question, only one specimen of *Protospirura* was found and that was *P. muris*. On the other hand, the known species of the genus *Protospirura* occur in rodents, and we have one case in which a

species of the genus *P. numidica*, first described by Seurat and Henry, from one of the wild Felidae, was subsequently (Seurat 1916) found in its normal host, a rodent. If the cat was the true host, it is still problematical as to whether the worm developed from one of the larvae from *Aphodius fimetarius* that had been fed to the cat, though the life history of other protospirurids would make this plausible. The larvae of *P. muris* have been reported from *Tenebrio* species by Leuckart (1867) and Marchi (1871) and from *Xenopsylla cheopis* by Johnston (1913). Seurat (1916) does not accept Johnston's finding, but regards *Mastophorus echiurus* Diesing, 1853, from the meal worm, as a larva of this worm. The species of the genus *Protospirura* may be distinguished as follows:

1. Six head papillae (apparently two of these are the lateral papillae, or what are termed amphids by Cobb, and these may be present, though as yet undescribed, in other species); male unknown... *P. labiodentata*.  
Four head papillae; male known.....2
2. Length of male over 30 mm.....3  
Length of male less than 30 mm.....4
3. Long spicule 1.2 to 1.4 mm.; short spicule 775 to 860 $\mu$ ..... *P. ascaroidea*.  
Long spicule 480 $\mu$ ; short spicule 350 $\mu$ ..... *P. muricola*.
4. Long spicule 830 $\mu$ ; short spicule 420 $\mu$ ..... *P. numidica*.  
Long spicule more than 1 mm.; short spicule more than 600 $\mu$ .....5
5. Maximum diameter of male 1 mm. or more; short spicule about 1 mm. long..... *P. muris*.  
Maximum diameter about 564 $\mu$ ; short spicule 622 $\mu$  long... *P. gracilis* sp.n.

#### ADDENDUM

Since the above manuscript was sent to press, Ortlepp (1924) has described a new species of *Protospirura*, *P. bonnei*, from the rat in Dutch Guiana. This species may be distinguished from those listed above in that the male does not exceed a length of 25 mm. and the spicules are short and equal in length (395 $\mu$ .)

#### 16 TO 1

Son: "Father, why do you insist that I study dentistry? I have told you several times that I want to be a doctor and specialize in eye treatment."

Father: "My son, do you realize that each person has 32 teeth, but only two eyes?"

#### OUT OF SIGHT

He: "Rather tough on a dog that is vaccinated. It can not talk about it with everyone it meets."

She: "The dog shouldn't object, however, the scar will not show."

## THE CAUSE OF FOOT-AND-MOUTH DISEASE DISCOVERED. THE FROSCH-DAHMEN BACILLUS

The 7th of April, 1924, was an honor day of the first rank for the German Veterinary Medical Research and especially so for the Hygienic Institute of the Veterinary High School of Berlin.

On that occasion the director of the Institute, Privy Counsellor Dr. Frosch, and the Division Chief of the Institute, Privy Counsellor Dr. Dahmen, announced before the Microbiological Association of Berlin and before many other guests the discovery of the causative agent of foot-and-mouth disease. The Veterinary Division of the Department of Agriculture of Germany was also represented by the Assistant Secretary Ramm, together with his staff; likewise the entire faculty of the Veterinary High School of Berlin led by the Dean was present on that occasion.

According to the statement of Dr. Frosch the discovery was made possible with the aid of the Kohler apparatus constructed by Zeiss which has already rendered such splendid service in the discovery of the cause of contagious pleuro-pneumonia. With short-wave ultra-violet rays of this apparatus, it is possible through photographic reproduction even at the highest magnifications to demonstrate the minutest particles and reproduce them in photograms which could not be attained by ordinary microscopical investigations. The tremendous difficulty of this work with the nerve-racking racket of Kohler's apparatus and the impossibility of focusing, necessitating many hundreds of photographs, should be only casually mentioned at this time.

Dahmen also succeeded in cultivating the causative agent on nutritive media of simple composition after he succeeded in separating the causative agent from the bactericidal action of the aphthous lymph. The more detailed description of the method will be published only after the Department of Agriculture, which contributed the funds for this investigation, has granted their consent for the same.

The inoculation of cultures of progressively higher generations into guinea pigs has produced a generally well known and recognized picture of guinea pig foot-and-mouth disease. However, the course of the infection was somewhat retarded when compared with the inoculations with fresh foot-and-mouth disease virus. The same results were also obtained in a cow which developed very pronounced salivation. In the photograms

taken with the aid of Kohler's apparatus Frosch recognized in the fluid of the vesicles of guinea pigs the etiological factor of foot-and-mouth disease in the form of fine rods.

The many photograms of the causative agent and cultures of agar and gelatin were demonstrated with the aid of lantern slides. The latter also were passed around in the lecture room of the Institute to the audience. On agar the causative agent forms a very fine film; single colonies are about 8 microns in diameter and the individual bacilli only 0.1 micron in size.

At the conclusion Frosch with his extreme modesty suggested designating the new organism, which he considers as a bacillus, with the name of *Löffleria Nevermanni*. However, in just recognition of the discoverers, the name "Frosch-Dahmen bacillus" would be more appropriate. Finally Frosch expressed his hope that with the aid of the new method it will be possible to discover other ultravisible organisms such as those of rinderpest, hog cholera, scarlet fever, measles, etc. (*Tierärztliche Rundschau*, April 20th, 1924.)

### STATE BOARD EXAMINATIONS

Nebraska	Capitol Bldg., Lincoln	June 4-5
	Supt. Dept. of Health and Welfare, Lincoln, Nebr.	
Colorado	Capitol Bldg., Denver	June 6-7
	Secretary, Dr. A. N. Carroll, 301 W. 3rd St., Pueblo, Colo.	
Tennessee	State Capitol, Nashville	June 11
	Secretary, Dr. M. Jacob, 312 W. Church Ave., Knoxville, Tenn.	
Arizona	State Capitol, Phoenix	June 15-16
	Secretary, Dr. Z. S. McNess, Glendale, Ariz.	
Washington	Pullman	June 16-17-18
	Secretary, Dr. A. R. Galbraith, Garfield, Wash.	
Mississippi	Jackson	June 17
	Secretary, Dr. Wm. L. Gates, Clarksdale, Miss.	
Massachusetts	State House, Boston	June 19-20
	Secretary, Dr. E. W. Babson, 343 Washington St., Gloucester, Mass.	
Arkansas	Old State House, Little Rock	June 20
	Secretary, Dr. J. H. Bux, Old State House, Little Rock, Ark.	
Pennsylvania	Vet. School, U. of P., Philadelphia	June 20-21
	Secretary, Dr. H. W. Barnard, 529 E. King St., Lancaster, Pa.	
North Carolina	Blowing Rock.	June 24
	Secretary, Dr. Wm. Moore, Raleigh, N. C.	
South Dakota	Pierre	June 24-25
	Secretary, Dr. Carle B. Lenker, Colome, So. Dak.	
Wisconsin	Capitol Bldg., Madison	June 24-25
	Secretary, Dr. T. H. Ferguson, Lake Geneva, Wis.	
Vermont	State House, Montpelier	June 25-26
	Secretary, Dr. Geo. Stephens, White River Junction, Vt.	
Maryland	Baltimore	June 26-27
	Secretary, Dr. Hulbert Young, 515 N. Charles St., Baltimore, Md.	

## CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

### RUPTURE OF THE DIAPHRAGM IN A COW

*By C. R. DONHAM, Corvallis, Ore.*

This is evidently quite an unusual condition and is therefore considered of sufficient importance to receive mention. It is interesting to consider the history and the conditions existing at the time the rupture is thought to have occurred, and the effects produced.

This was a pure-bred Holstein cow, seven and a half years old. She was a high-producing animal, having an official yearly record of 18,707 pounds of milk with 834.7 pounds of eighty percent butter. She had been a strong reactor to the agglutination test for infectious abortion for five years but had never aborted. There were five normal pregnancies with no difficulty in breeding except just prior to the last pregnancy when the animal was bred nine times.

The rupture of the diaphragm is thought to have occurred at the last parturition. On September 10, 1923, she gave birth to a heifer calf weighing 110 pounds. There was no difficulty in parturition and the fetal membranes were expelled normally. The udder development of this animal was excessive and following calving it became swollen and edematous, and there were symptoms of developing parturient paresis. In the evening of September 11, 1923, we were called and found the conditions described but did not consider it necessary to inflate the udder. A heavy canvas suspension apparatus was applied to the udder with the idea of reducing the edema and also perhaps to apply enough pressure to have some beneficial effect in preventing parturient paresis. (The application of pressure to the udder of a cow, in this manner, to prevent or alleviate symptoms of milk fever, is entirely in the experimental stage so far as we are concerned.) The following morning the edema of the udder had been materially reduced and the animal was apparently in good health except for a passive congestion of the udder. Following calving, the milk production was normal for about one month,

after which a chronic mammitis developed which decreased the flow of milk and rendered it unfit for use.

During the following weeks the animal decreased in flesh very rapidly without any apparent reason, the milk flow became greatly reduced and characteristic of garget, and there were evidences of serious ovarian disturbances. Consequently, the animal was considered unprofitable to retain in the herd and was sold to be used in the dissecting laboratory. The animal was killed on December 4, 1923.



FIG. 1. Hernia of the diaphragm.

The autopsy report includes some pathology which will not be mentioned here because it has no relationship to the rupture of the diaphragm. The accompanying photograph, furnished by Dr. F. W. Miller, illustrates the rent in the diaphragm. A cross-section of the liver is shown covered over by the right crus of the diaphragm, which is somewhat curled, due to drying. A mass of the liver tissue is shown protruding through the diaphragm into the thoracic cavity. The location of the rupture of the diaphragm was about four inches to the right of the median plane and just above the level of the posterior vena cava. The opening in the diaphragm was a separation of the fibrous tissue along its long axis, without any cross tearing of the tissue. The protruding liver tissue was pedunculated, with a fibrous formation around the neck of it, indicating that the condition had been present for a considerable length of time. There was evidence of extensive injury in both the anterior part of the abdominal cavity and in the thoracic cavity, due to foreign bodies having passed out from the reticulum. There were extensive adhesions and hemorrhage in both cavities. There was an exceptionally

large amount of foreign material present in the reticulum and some in the abomasum.

All of the above mentioned pathology is thought to have been produced at the last act of parturition which was approximately two and one-half months before the animal was destroyed. This conclusion is based upon the apparent age of the various lesions mentioned. It is interesting to consider the life of the animal since the last calving. It will be noted that the production and other body functions were evidently normal for about one month. The relationship between the symptoms produced and the lesions found will be left entirely with the reader.

### LAMINITIS, OR WHAT?

*By F. W. CRAWFORD, Big Fork, Ark.*

While on a vacation and visiting here in Arkansas, and although not soliciting any practice, I have been frequently called upon, more to pass my opinion and give advice, than for any other reason, as a veterinarian in this immediate neighborhood is rather a novelty, and a "dog doctor" is beyond all imagination. Every man here is his own doctor, curing anything and everything, with such common drugs as hickory bark, pine resin and tobacco juice, to the up-to-date preparations of "Watkins," "Rawleighs," or some such liniments, equally valuable if given internally or externally. However, we all live to learn, and here is an interesting case that I was allowed to see, and thought it might prove of interest in your "Clinical and Case Reports."

*Subject:* Bay mare, 7 years old, about 1300 pounds, always had been healthy, not subject to any chronic ailments, never had been with foal. Used for general farm work and logging.

*History:* On March 13, there was a noticeable diarrhea, but the animal was worked (logging) all day. No other symptoms or ailments were apparent and animal was reported having a good appetite, plenty of life and vitality. That night, after the evening meal, she was apparently in pain; groaning, lying down, getting up, etc., but there was no rolling or violent symptoms, and no sweating was noticed. The diarrhea was copious and very watery. The owner became concerned and gave her various doses of patent colic remedies. He was up with her most of the night. The following morning he thought she was

going to die, so called me. He had heard that I was "kinda of a hoss doctor."

*Symptoms:* The mare was standing with her head hanging very low, feet bunched under, but with the hind feet rather far apart. Although in great pain and very depressed, she did not act violently. She had lost greatly in flesh and seemed very weak—rather stiff and disinclined to move—the feet seemed very sore. Peristaltic movement was loud and violent. Very watery and violent stools—neither bloody nor fetid. Urine—normal in passage, frequency, amount and consistency. Respirations, besides the grunting, were normal. Temperature 102° F. Pulse imperceptible, heart very slow and weak. Mucous membranes very slightly congested. She desired water continuously, and even showed a ravenous appetite.

*Diagnosis:* Botulism. I knew in my own mind that this was far from it, as I have had several experiences with botulism. I also happened to know that the owner suspected a neighbor as having given poison, so the diagnosis satisfied his poisoning impulse, but eliminated the neighbor. It also stumped the "old timers" who were hanging about, as it named something practically foreign to them. So everyone was satisfied with the diagnosis, except myself. Personally I suspected either "acute, infectious diarrhea" or "founder." I could not get the symptoms to fit the former, nor yet the latter. The history also contradicted the latter. The team-mate of this animal had been constantly with her under the same working and feeding conditions, and showed no unusual symptoms of any kind. I personally examined all the feed, which consisted of corn chops, bran, home-grown and cured prairie and oat hay. It all seemed in excellent condition to me. The owner was confident that the animal had not had access to more than her regular feed. I also investigated and inquired into the watering of these animals and could find nothing out of the way.

*Prognosis:* Withheld.

*Treatment:* I knew the animal would not live through the day without some kind of stimulant. I immediately gave a subcutaneous injection of  $\frac{1}{4}$  gr. strychnin, and two additional doses at one-hour intervals, until the pulse was strong and steady. As I was not here to practice, my drug supply was both meager and inadequate, but I finally concocted the following: Fldest. nux vomica and gelsemium, one dram each, and mucoseptone (Jen-Sal), two drams, to be given diluted in a

quart of water, as a drench, every two hours. I also proceeded to flush out the rectum and give a high enema of lukewarm salt water. I ordered the animal blanketed and to be kept warm and dry, as the weather was cold and snowy. Food to be withheld—water, warm and in very limited amounts.

That night the owner could not see any change for the better, but the heart was still in good shape. Peristalsis was not so pronounced and the animal seemed much more at ease, so I encouraged the owner, and he gave the medicine every two hours all night.

The next morning he still had his doubts, although the diarrhea was checking, the peristaltic movement almost back to normal and on general appearance the animal gave a favorable prognosis. She seemed very sore and stiff, especially her feet, and was loath to move about, although she would occasionally lie down, only to rise painfully again. I replenished his supply of medicine (draining mine), with the same orders as the day before, except a slight increase in water and allowed some warm, bran mash.

The morning of the 16th there was some improvement. The same treatment was maintained, but with doses every four hours, and more food and water allowed. On the 17th there was a noticeable improvement. On the 18th the animal could walk about without any great amount of stiffness or pain. She was very gaunt and had lost considerably in flesh. We had run out of medicine, but, in the meantime, by sending to a neighboring town, some thirty miles distant, I had obtained some drugs and compounded a tonic of Carlsbad salts, powdered nux and gentian. The horse was put on this, and is now on a good recovery, no symptoms of anything remaining.

What diagnosis could be rightly applied to this condition—and what was the cause of it?

### HYDROPS CYSTIDIS FELLAE IN A COW<sup>1</sup>

*By FRED BOERNER, JR., Philadelphia, Pa.*

The specimen consisted of a gall-bladder from a cow, slaughtered in one of the slaughter houses in Philadelphia, Pa. The animal appeared normal before slaughter, and on post-mortem nothing abnormal was observed except a peculiar appearance of the gall-bladder. The liver appeared normal,

<sup>1</sup>Contribution from the Bureau of Animal Industry of the Pennsylvania Department of Agriculture. New Series No. 18.

and was passed for food after the removal of the gall-bladder. The latter was normal in size, walls thickened and, instead of appearing greenish, due to distention with bile, it was white, fully distended and had the general appearance of a cyst. Upon

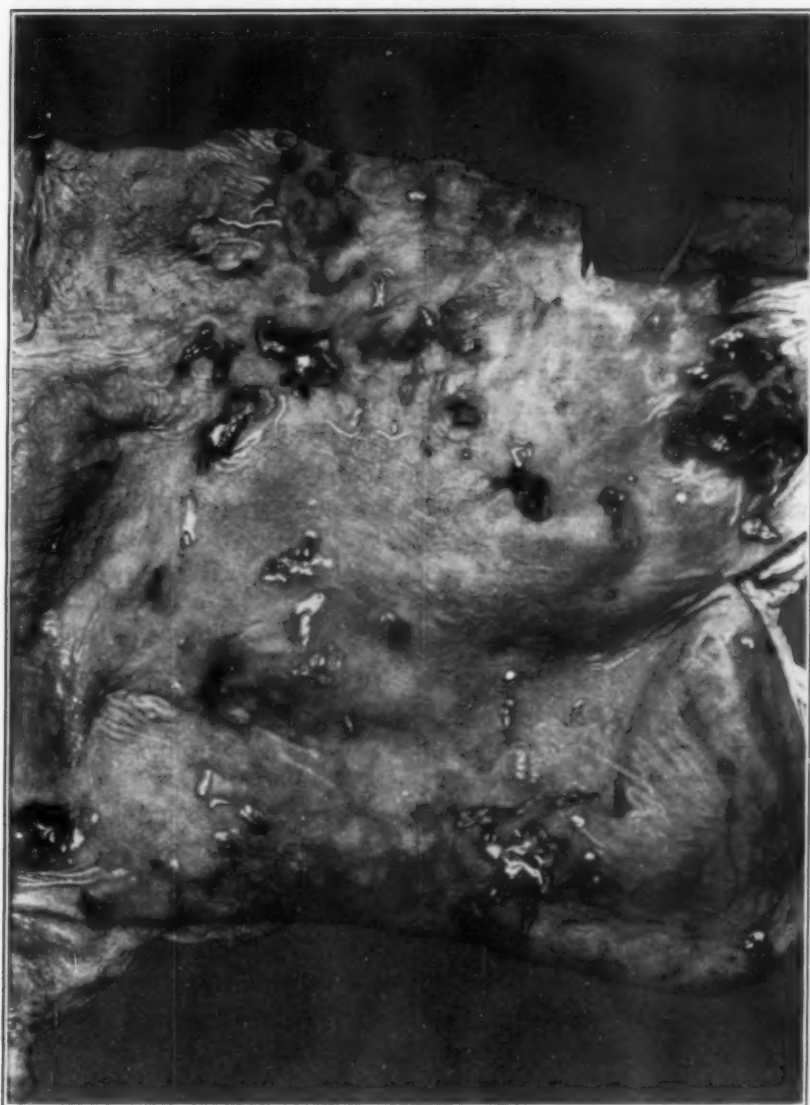


FIG. 1. Hydrops Cystidis Felleae.

pressure, the contents could not be forced out through either duct. Upon opening, the content was found to be a perfectly clear, colorless fluid, which proved to be sterile. No traces of bile

could be demonstrated by chemical tests. The fluid was very rich in albumin. The walls of the bladder were thickened, and the mucous membrane was very smooth and white in color. Scattered over the surface were numerous atheromatous areas and calcified plaques. In general, it had about the same gross appearance as the intima of an atheromatous aorta. Sections showed the lining membrane entirely devoid of epithelium and replaced with dense connective tissue, showing small and large areas in various stages of degeneration and calcification.

The pathogenesis of the case was probably as follows: The condition began as an acute cholangitis, which became chronic, resulting in the occlusion of the cystic duct. This prevented the flow of bile into and out of the gall-bladder. The bile was absorbed and replaced with a clear fluid, either secreted by the mucous membrane or of the nature of an inflammatory serum. Chronic degenerative changes then followed in the wall of the bladder, resulting in the condition noted on post-mortem.

Although we were unable in this case to show the communication between the cystic and common duct, as the bladder was removed when received, there is no doubt that such a communication was established.

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### WOOLLY-POD MILKWEED POISONS CATTLE AND SHEEP

Woolly-pod milkweed, a plant growing rather abundantly in parts of western California, has long been suspected by stockmen of being poisonous to cattle and sheep. Recent investigations by the United States Department of Agriculture have shown that it is very poisonous to these animals. Although no cases have been reported, it is thought that it may also be poisonous to horses. Animals are not likely to eat it except when other forage is scarce, but it is particularly dangerous, as only a small quantity is necessary and the dried plant may be eaten in hay. As little as 0.1 of a pound per hundredweight of animal may poison and 0.22 of a pound of the green material, or its equivalent of dried plant per hundredweight, may cause death.

A detailed account of the experimental work and descriptions of the plant with photographs have been published in Department Bulletin 1212, "The Woolly-Pod Milkweed as a Poisonous Plant," by C. D. Marsh and A. B. Clawson. Copies of the bulletin may be obtained, as long as the supply lasts, by writing to the Department of Agriculture, Washington, D. C.

## REVIEW

MANUEL DE MEDECINE LEGALE VETERINAIRE (Manual of Veterinary Legal Medicine). By Albert Lhoste, Director of the Abattoirs of Mans. viii + 96 pages. Published by Vigot Freres, Paris, 1924. Price, 5 francs, paper.

M. Lhoste has prepared this manual as a guide for veterinarians called upon to testify as experts in medico-legal cases. As stated in the preface, by Prof. A. Daille, no attempt has been made to prepare a treatise on the subject, but rather nothing more than a vade-mecum for practitioners.

Chapters are devoted to (1) general considerations of expert testimony, (2) responsibility of experts, (3) their remuneration, (4) reports, (5) certificates, (6) signs of death, (5) ante- and post-mortem lesions, (8) death by starvation, (9) death from freezing, (1) death from insolation and heat-stroke, (11) asphyxia, (12) poisonings, (13) wounds, (14) wounds from falling, being crushed, and railway accidents, (15) burns, (16) lightning stroke, (17) scars, (18) signs of pregnancy, (19) duration of gestation, (20) signs of recent parturition, (21) characters of the fetus at different periods of intra-uterine life, (22) extra-uterine appearance of the new-born, (23) bestiality, and (24) a glossary of terms used in medico-legal practice.

On the whole, a handy little book.

## ABSTRACT

ENCEPHALITOOZON RABIEI, Parasite of Rabies. Y. Manouelian and J. Viala. (Transmitted by Roux.) *Compt. Rend. de l'Acad. des Sci., Paris* (Jan. 14, 1924), 178, p. 344.

Our studies on street rabies in the dog, man, the rabbit, the monkey (macaque), and on fixed-virus rabies in the rabbit, have revealed throughout the cerebrospinal axis, in the sympathetic nervous system, in the peripheral nerves, and in the salivary glands, peculiar formations which we consider as the parasite of rabies.

They consist of elongated, fusiform, piriform or shuttle-shaped corpuscles, averaging from 1 to 2 microns. Their protoplasmic body appears to enclose granulations of chromatin and seems surrounded by a membrane. They present sometimes a clear space at one of the extremities.

They are not stained by the methods of Ramon y Cajal, Levaditi, or ourselves.

These corpuscles are found in more or less considerable numbers in the cytoplasm of the nerve cells and in the cytoplasmic prolongations of the neurons. Outside of these cells they are found disseminated in the nerve tissue.

We have also discovered them in abundance in the cells of the acini of the salivary glands and in the lumen of the excretory canals, consequently in the saliva.

These parasites are certainly very similar to the corpuscles demonstrated by Doerr and Zdansky in the sections of Kling, which came from brains of rabbits inoculated with Swedish encephalitic virus. They are very similar also to those recently described by Levaditi and Nicolau, in the brains of rabbits inoculated with the virus of Kling, Thalimer, and Bull, Oliver and Twort.

Levaditi and Nicolau think that the corpuscles of the encephalitis of the rabbit are protozoan parasites, perhaps of the group of microsporidia. They have named them *Encephalitozoon cuniculi*. As we believe the parasite of rabies to be very similar, let us repeat, to *Encephalitozoon cuniculi*, we propose to name it *Encephalitozoon rabiei*.<sup>1</sup>

What is the relation between the corpuscles of Negri and these forms? We believe that the corpuscles of Negri are the product of degeneration of the parasite at the interior of the nerve cell: the parasites invade the cell and multiply there. We have been able to demonstrate their agglutination in mass, especially in the nerve cells of the horn of Ammon. Following this agglutination there is a coalescence of the parasites; and it is at the expense of these masses of parasitic origin that the bodies of Negri are constituted.

The question arises as to a particular reaction in the nerve cell, since in these cells of the salivary glands, where the parasites multiply, the bodies of Negri never exist; however, in the intraglandular nerve ganglions we pointed out a long time ago the presence of the corpuscles of Negri.

A further interesting fact is that in fixed-virus rabies the corpuscles of Negri are small and very rare. It is understood that a virus exalted by multiple passages does not give such strong reactions in the nerve cell as in street rabies.

J. R. M.

<sup>1</sup>In a note which appeared in the Comptes Rendus of January 7 last, Levaditi and Nicolau put forth the hypothesis that the parasite of rabies is of the same nature as that of natural encephalitis of the rabbit.

## AMERICAN VETERINARY MEDICAL ASSOCIATION

Proceedings of Sixtieth Annual Meeting, Montreal,  
Canada. August 27 to 31, 1923.

*(Continued from p. 253, May Journal)*

### TUESDAY MORNING SESSION

#### (SECTION ON SANITARY SCIENCE AND POLICE)

August 28, 1923

The first session of the Section on Sanitary Science and Police convened at 10:25 a.m., Dr. R. C. Reed, of College Park, Md., presiding.

CHAIRMAN REED: Gentlemen, if you will please come to order, we will open the meeting. Inasmuch as it is so late, I am sure you will be glad to hear that the chairman of your section is not going to make an address.

In opening this session, I want to say that I am afraid we do not always keep in mind the main object of this particular line of work. The name of the section is rather old, and possibly does not apply quite as well as it did earlier. "Sanitary Science and Police" has rather a legal sound, however, and is not so bad a name.

The object of those doing work along these lines is to recognize the presence of infectious disease at the earliest possible moment and check its spread and thus eradicate it; that is, it is not primarily control but eradication that we want.

State and Government officials cannot do this work alone. The remark was aptly made only last evening by an eminent veterinarian: "It is safe to say that the official practically never discovers an infectious disease; it is the veterinarian in the field who does that for us, and the official simply asks to step in and help, in his official capacity, to check and eradicate that disease by legal means with the help of the local veterinarian." Therefore, it is up to the practicing veterinarian to be able to recognize these diseases; to be, we might say in slang, "on the job" enough to report them promptly, and to get the help that is necessary to control them.

The Secretary of this Association once made the remark that the object of this Section is to discuss problems that are of interest to such a body as the United States Live Stock Sanitary

Association, and furthermore, to report to them suggestions and methods of controlling disease. That is again the official phase of the work.

But let me repeat that it is, after all, the veterinarian who has to be on the job constantly. He is the one at the foundation of this control and eradication work. The veterinarian must then show himself capable; he must show himself better fitted than the live stock owner to cope with these problems, both for his own success as a practitioner and as a sanitarian. It is up to those of us in official positions to help the practitioner to keep up to standard so that he may help us to do our official work.

The next item on the program is the report of the secretary, Dr. Hall. (Applause.)

Dr. Orlan Hall presented his report. (Applause.)

#### REPORT OF THE SECRETARY

##### SECTION ON SANITARY SCIENCE AND POLICE

The duties of a section secretary are to arrange the program of the section in cooperation with the chairman, the major portion of his duties being the securing of suitable papers to be presented before the section during the annual meeting. In order to give you some idea of the arrangements which were made in preparing our program, I would point out that during the meeting of the U. S. Live Stock Sanitary Association, held in Chicago, December last, four of the six section officers were present. Secretary Hoskins was also present, and called a short, informal meeting in conjunction with these officers.

I regret that I was not able to attend this meeting, but, due to circumstances over which I had no control, I was compelled to cancel my trip to Chicago. However, I am glad to say that our chairman was there to represent this section, and has conveyed an account of the meeting to me. It was suggested, at that meeting, that three papers be presented at each of the half-day sessions. This procedure will allow for no crowding of the program, and will give ample opportunity for a thorough discussion of each subject.

An effort has been made to procure papers on subjects calling for sanitary control measures. We have endeavored to keep from infringing on the rights of the other two sections, namely, "Education and Research" and "General Practice." Authors have been duly informed as to subject-matter, and length of their papers. All of the correspondence which has passed be-

tween the members of this Association and your secretary is available at this meeting, and can be read in whole or in part, if so desired.

For the benefit of the incoming secretary, I would like to suggest to the members of this Association who are interested in sanitary control measures, that early in the year, before the 1924 meeting is held, they communicate with the secretary, informing him as to whether or not they have papers which they desire to present before this section. This would relieve the chairman and secretary to a great extent in preparing the program.

To illustrate the point, a member may be requested to present a paper on a certain subject, but, not being in possession of sufficient data, or not being able to attend the meeting, he declines. Therefore, the secretary necessarily is obliged to carry on considerable correspondence with other members of the Association, endeavoring to locate the member who has the necessary data at his disposal, and is in a position to attend the next meeting. I leave this suggestion for your consideration.

I take this opportunity of thanking those members who are to present papers before the section. They have cooperated with your secretary, and I can assure you their cooperation has been appreciated.

ORLAN HALL, *Secretary.*

CHAIRMAN REED: Gentlemen, have you any additions or corrections or suggestions regarding the Secretary's report? If not, we will pass on to the reading of the papers, the first one on the program being "The Serum Therapy of Glanders, with Special Reference to Glanders in Man," by Dr. E. A. Watson, Health of Animals Branch, Department of Agriculture, Ottawa, Canada. (Applause.)

Dr. Watson read his paper. (Published in the JOURNAL, November, 1923.)

CHAIRMAN REED: If there is no further discussion, we will pass on to the second paper of the morning, "Excessive Pig Losses Can be Prevented," by Dr. A. T. Kinsley, Kansas City, Missouri. (Applause.)

Dr. Kinsley read his paper. (Published in the JOURNAL, November, 1923.)

CHAIRMAN REED: The next paper on the program is "The Control of Internal Parasites of Swine," by Dr. Maurice C.

Hall, Zoological Division, Bureau of Animal Industry, Washington, D. C. (Applause.)

DR. MAURICE C. HALL: This film, "Exit Ascaris," is one that probably most of you have seen at least once; some of you have undoubtedly seen it a number of times, but I don't think it will bore you to see it again. Furthermore, what I have to say will be substantially what Dr. Kinsley and what Dr. Gibson have said. I am more or less in sympathy with Dr. Atherton's ideas, though I haven't any figures to check Dr. Atherton, and, as we usually disagree when we get together, I really should wait until I have something on which to base a disagreement with him.

I saw Dr. Kinsley last night, and suggested that while he was making his talk, he say just what I would want to say anyway, so that all that would be necessary for me to do would be to say, in the language of some Western character, "Them's my sentiments!" Most of what Dr. Kinsley has said is precisely what I would say, but from his long experience in swine practice, he has said much that I would not be able to say, and he told you a good deal that I don't know.

(The film, "Exit Ascaris," of the U. S. Department of Agriculture, was shown)

What I expected to emphasize today was the importance of parasites in the production of diseases of young animals, more especially, of course, in swine. Dr. Gibson has already emphasized that.

About the time of the International Live Stock Show at Chicago, Dr. Buckley and I, on the way to that show, were discussing the matter of infant mortality in live stock, and it seemed to both of us that it was a subject that had received almost no appreciation. I haven't any technical material to present today; I don't care to discuss the results of any experiments or give you any new findings. As Dr. Kinsley has said, this subject is one which is so simple and elementary, so familiar to all of you, that it may sound a little like a high school proposition. However, in the case of the human infant it has only been in recent years that we have come to regard a high infant mortality as an inexcusable and preventable thing. We have not yet begun to appreciate that in the live stock industry a high infant mortality is also an inexcusable and preventable thing.

It was within a hundred years not an uncommon thing for a family to have a large number of children and lose half of them

in infancy or early childhood. Today, under better conditions in a civilized country, that has become an unusual thing, and our practice as regards the feeding of children and the sanitary surroundings for children has resulted in markedly decreasing that infant mortality.

In the case of live stock, we now have evidence of an incontrovertible sort that infant mortality, at least among swine, may be largely prevented by sanitation. We have been preaching sanitation on general grounds for many years as a preventive measure in the case of animal diseases, but experience shows that people do not do things on general principles; they do things for a specific purpose.

The particular purpose for which this sanitary system has been adopted has been for the prevention of worm infections in young pigs—and here we have in this film some evidence which is conclusive. As Dr. Gibson has said, the farmer is a man who must be shown. This system has worked for three years in McLean County, Illinois, and the farmer is enthusiastic about it; he is convinced. The system is spreading through adjacent counties, and is being used through various parts of the United States. It may need modification to suit local conditions outside of the Middle West, but in the main these principles of sanitation will probably apply everywhere.

Now, if you will run the film, doctor, we will let it tell its own story.

(The first reel was shown)

This film, by the way, is the property of the Canadian Government and is loaned us through their courtesy for use here today.

That is my immediate Chief and personal friend, Dr. Ransom. Dr. Ransom is, in my opinion, next to Dr. Railliet, the best veterinary parasitologist in the world; a man who has a very wide and sound knowledge of parasitology, and whose contributions to veterinary parasitology and to scientific research in parasitology in America have been of great importance. I believe, as Dr. Railliet is a man over seventy and Dr. Ransom is just over forty, that we may readily expect Dr. Ransom's achievements to equal in time those of Dr. Railliet.

So much of the reel, of course, presents the problem, and the second reel indicates a solution for this condition. I might say just in passing, while we are waiting for this reel, that our care of the young pig or of live stock in general may have to

start a generation before the animal is born. We know that in the case of certain parasitic worms it is possible for the young animal to become infected in the maternal uterus. That is true of lung worms; it has been shown in Germany and in our laboratory that it is true of at least one of the dog ascarids, and it is theoretically possible with any number of these worms which have the habit of migrating from the intestine through the tissues and returning to the intestines or other parts of the digestive tract to complete the development to the adult worm.

It appears entirely probable that many of the cases of severe ascarid infestation in pups originate with an infection during uterine life, and a study of the number of worms present in pups shows that they are at a maximum in early life and later diminish in number. In fact, that is generally true for parasitic worms, that young animals are more susceptible to them.

(The next reel was shown)

It is getting rather late so I want to recapitulate briefly the general ideas which have already been expressed. As I said, there is some danger of intrauterine infestation with worms, but for the present our problem concerns itself for the most part with the young animal at the time it is born. Up to that time it is comparatively secure in the maternal uterus, comparatively safe from traumatic injury and infection with germs and worms.

At the time of birth, this young animal is thrown out on a cold, hard world. Under farm conditions, such as were shown you in the first part of this film, this young animal arrives in the midst of filth and grows up in the midst of filth. Keep in mind that manure carries pathogenic bacteria and the eggs and larvae of most of the parasitic worms. Some of them, such as the kidney-worm, pass in the urine. But for practical purposes if you regard the manure as dangerous and take measures accordingly, you will cover most of these cases.

Between this young and tender animal which has not yet developed any immunity, an animal whose tissues are tender, and the mature, profitable animal to which the farmer looks for his income, there is a barrier on most farms in the form of filth, with all that that implies in pathogenic bacteria and worm infestations.

It is the business of the farmer to develop those sanitary measures, or at least to apply them, which shall build a road across that barrier and lead these young animals from infancy into profitable maturity.

In that connection the veterinarian has an advisory capacity. Sanitation means a bigger, better and more profitable live stock industry, and that means business and benefits for the practicing veterinarian. Unless those sanitary measures are applied, the usual result will be an effective barrier, except in the cases where good fortune or considerable individual resistance carries an animal on to that profitable maturity which is the logical goal of our live stock.

There has been very much indifference in the live stock industry to the death of the young animal, just as there was too much disposition at one time to take the death of the baby as the dispensation of what has been called a wise and unscrupulous Providence. No death of a young animal should go unchallenged. There is always a reason why the young animal dies, and we must combat, wherever we meet it, this idea that this animal never amounted to anything anyway. To be sure, this young animal has not built up a reputation for itself—it couldn't. But we have got to protect this young animal, and realize that if it dies, some cause has entered in, and that in many cases that cause could have been taken care of and the death prevented.

There is no need of saying any more to you. This sounds like high school stuff because you all know it. I am not trying to tell it to you because you don't know it, but I think we all need to evaluate it higher, to appreciate it more, and to act accordingly and not allow this matter to be neglected.

You veterinarians are in contact with the live stock industry and can do a tremendously good work. You can realize that these young animals are much more susceptible to parasites than our older animals. Chicks have gapeworms, but older chickens do not. It is your calf and lamb and colt that die of lung worms and stomach worms and other worms. The same number of parasites injures them more than it would the older animal, and they usually have more parasites. Many of these older animals cannot be infected experimentally. We learned long ago in our laboratories that if we wanted to develop worms in experiment animals, we must pick young animals. Our experiments fail too often with older animals, and in some cases will fail almost uniformly with old animals.

I think the idea has been emphasized to you sufficiently today, but I trust that we shall all take this matter to heart and act accordingly. I have been talking this and writing it for the last

six or eight months, and I want to talk it and write it until it is evident that it is generally known and appreciated, and if necessary to the point of saturation where people say, "Well, there's no use hearing this man talk: we know what he's going to say." Let us all talk it and practice it! It really means a tremendous lot to the live stock industry. I thank you. (Applause.)

CHAIRMAN REED: This paper is open for discussion; or shall we go on with Dr. Atherton's paper and continue the discussion after that? If any one has anything special to bring up, in connection with Dr. Hall's paper, we would be glad to have it now. If not, we will listen to Dr. Atherton's paper on "Hog Cholera Control vs. Prevention."

. . . Dr. Atherton presented his paper. . . (Applause.)  
(Published in the JOURNAL, December, 1923.)

CHAIRMAN REED: If there is nothing more to come before this session, we will stand adjourned until tomorrow morning.

. . . The meeting adjourned at 12:50 p.m. . . .

#### ADJOURNMENT

#### FRIDAY MORNING SESSION

August 31, 1923

The meeting convened at 9:40 a.m., Dr. R. C. Reed presiding.

CHAIRMAN REED: Gentlemen, will you please come to order? While we have rather a short-appearing program, yet it is decidedly long if we consider some of the questions involved.

The first paper on the program is entitled "A New Disease of Cattle Simulating Hemorrhagic Septicemia and Blackleg, Due to Damaged Sweet Clover," by Dr. Frank W. Schofield, Ontario Veterinary College, Guelph, Ontario. Dr. Schofield. (Applause.)

DR. FRANK W. SCHOFIELD: I don't know how many of you are familiar with sweet clover, so going up the mountain yesterday, I plucked a little, but it is unfortunately very withered at the present time. This plant, sweet clover, has been known in Europe for about two thousand years, growing around the Mediterranean basin, but only for two hundred years has it been growing in America. It most likely came in accidentally. The plant is easily distinguished from other plants. When it is young, it looks quite a little like alfalfa, but as it becomes more mature, the stalk develops very markedly, and also the stalk is hollow but rapidly develops a large quantity of cellulose,

it becomes very tough and fibrous and indigestible. When in blossom, there is that very definite perfume of the sweet clover; then the leaf is very bitter to the taste, indeed. There are two things I would like you to remember with regard to the plant. One is the heavy development of the stalk; much more so than any of the other legumes. For instance, here is a little bundle of alfalfa stalks from mature alfalfa and there are stalks from immature sweet clover—a marked difference, and the stalks are hollow. There are a variety of species of sweet clover, but the one that is most commonly being grown by the farmers is *Melilotus alba*, the white sweet clover. The other varieties are slightly different botanically. The yellow variety does not grow to the same height; it does not become so bushy as the white sweet clover, and that is more popular in some districts. Of course, there is this difference in species to be considered in dealing with sweet clover as a disease-producing plant.

Until recently, sweet clover was considered to be a weed quite generally, and a noxious weed, and certain government institutions had proclaimed against the cultivation of sweet clover, considering it as essentially a weed, but some farmers persisted in growing sweet clover because it had been grown for centuries in Europe and in other countries. In China they grow it for the making of baskets, and so on. So they grew this material and fed it to cattle; made hay out of it; plowed it under, and the farmers who worked with it finally convinced others that sweet clover was something other than a weed, and then a regular boom came for sweet clover and everybody was growing it, especially during the war when the sweet clover seed was fetching a tremendous price.

It unquestionably is a most important plant, because of certain things that we will take up later in connection with the plant destroying stock, because of something that occurs in it. We cannot discard this sweet clover plant; it is a plant of considerable economic significance. First of all, it will grow almost anywhere, on the poorest of soils; on a white, sandy soil which would grow practically nothing else, sweet clover will grow and rapidly improve that soil.

Then there is no plant that can compare with sweet clover as a crop fertilizer, for plowing the sweet clover under and improving the humus in the soil and the chemical composition of the soil. In other words, there is no plant to compare with sweet clover for improving the soil both physically and chemi-

cally. Then it produces good pasture, and also it can be made into ensilage and into sweet hay. With regard to ensilage, I would not say it compares favorably with corn, except in this respect: that sweet clover does not require a great deal of handling. The stuff is sown, cut, and put into the silo; whereas in the case of corn there is a great deal of manual labor in keeping the corn clean. Sweet clover is ready for the silo at a time when corn is not. There are a great many things to be said in favor of sweet clover as a valuable farm product.

Dr. Schofield then presented his paper. (Applause.) (Published in the JOURNAL, February, 1924.)

CHAIRMAN REED: We will pass on to the next paper on the program, "No-Lesion, Tuberculin-Reacting Cattle," by Dr. E. C. Schroeder, Bureau of Animal Industry Experiment Station, Bethesda, Maryland. (Applause.) Dr. Schroeder presented his paper. (Applause.) (Published in the JOURNAL, February, 1924.)

CHAIRMAN REED: We still have some matters to take up. First, is there any matter that any one has to bring up before this section before we take up the election of section officers? Is there any new or unfinished business? If not, we will proceed to the election of section officers. Nominations are in order. We have to elect a Chairman and a Secretary. Nominations for Chairman of the section for the ensuing year are now in order.

DR. CAMERON: I move that the present Chairman be again nominated for the position.

CHAIRMAN REED: I feel that that is hardly just. The present Chairman has held the chair down for two years, and that is almost an unprecedented thing, as I understand it. I think somebody else should be nominated for that office. It seems to me that the present Secretary is a good candidate for that office and would be the logical man to fill it.

DR. C. A. MITCHELL: I nominate Dr. Orland Hall.

CHAIRMAN REED: Dr. Orland Hall has been nominated for the office of Chairman of this section. If there are no further nominations, a motion to close the nominations is in order.

DR. E. C. SCHROEDER: I move that nominations be closed.

. . . The motion was seconded by Dr. Allen and carried . . .

CHAIRMAN REED: How shall we proceed to the election? It is supposed to be by ballot.

DR. J. A. ALLEN: I move that a standing vote be taken.

The motion was seconded by Dr. Cameron and carried . . .

CHAIRMAN REED: The motion having been unanimously carried, I declare Dr. Orland Hall duly elected as Chairman of the section, for the ensuing year.

Now we must have a Secretary.

DR. J. A. KIERNAN: Mr. Chairman, I nominate Dr. J. H. McNeil, of New Jersey, for Secretary of this section.

CHAIRMAN REED: Dr. J. H. McNeil has been nominated for Secretary of this section during the next year.

. . . The nomination was seconded by Dr. Hargrave . . .

CHAIRMAN REED: Are there any further nominations? If not, we will declare the nominations closed, Dr. McNeil being the only candidate.

DR. KIERNAN: I move that the Secretary be instructed to cast the unanimous vote for Dr. McNeil.

SECRETARY HALL: I have cast the unanimous vote of the Association for the election of Dr. McNeil as Secretary of this section during the ensuing year.

CHAIRMAN REED: I declare the election of Dr. McNeil as Secretary of this section for next year.

That concludes the business that is to come before this section this morning, unless there is something further to bring up. If not, we will stand adjourned until next year.

. . . The meeting adjourned at 11:30 a.m. . . .

#### ADJOURNMENT

#### "CLOVER" IS DEAD

"Clover," the world's oldest horse, died recently in his 53rd year. He was born June 15, 1871, and owned by Rev. Uriah Myers, of Catawissa, Pa. Two years ago, "Clover" was exhibited at Madison Square Garden. The New York Jockey Club recently voted him a pension. "Clover" was fifteen and one-half hands high, trotting-bred, and had a full mane and all of his teeth. Mrs. Warren G. Harding sent a check for \$100 to the Rev. Mr. Myers, a short time ago, so that "Clover" might be assured all the comforts of life during his old age.

The body will be mounted in the American Museum of Natural History, in New York City.

# NEW MEMBERS OF THE A. V. M. A. ADMITTED AT THE MONTREAL MEETING, AUGUST 27-31, 1923

- Agnew, T. H., Pasadena, Calif.  
 Armstrong, J. H. O., Montreal, Que.  
 Atkinson, Chas. H., Fresno, Calif.  
 Baker, Chas. B., Montreal, Que.  
 Baker, F. H., Gardnerville, Nev.  
 Baker, Frank J., Gouverneur, N. Y.  
 Ball, Robert A., Modesto, Calif.  
 Baughman, D. E., Fort Dodge, Iowa  
 Beaver, Norman, Hull, Que.  
 Beck, Francis W., Jacksonville, Fla.  
 Bedard, Joseph A. E., Quebec, Que.  
 Bedard, Jean M. F., Limoilou, Que.  
 Beggs, S. W., Lamar, Colo.  
 Beltran, Luis A., Havana, Cuba.  
 Bennett, John E., Calgary, Alta.  
 Bolton, John A., Downey, Calif.  
 Bishop, Chas. P., Sunbury, Pa.  
 Bishop, H. H., Toronto, Ont.  
 Bitler, Sherman E., Turbotville, Pa.  
 Bittles, E. E., Waterford, Pa.  
 Blatchford, Robert F., Detroit, Mich.  
 Boast, Chas. R., Montreal, Que.  
 Booth, Elias T., Philadelphia, Pa.  
 Brennan, W. P. B., Quebec, Que.  
 Brink, J. H., Owego, N. Y.  
 Broadhurst, Roy W., Long Beach, Cal.  
 Brockmeier, Elmer A., Mapleton, Minn.  
 Brower, Wm. P., Kanawha, Iowa  
 Brown, J. Howard, Rich Square, N. C.  
 Brown, Lester H., Elkhorn, Nebr.  
 Brunett, Earl L., Ithaca, N. Y.  
 Bryan, E. W., Athens, Ala.  
 Bullard, John F., Manhattan, Kans.  
 Burk, James A., Shippensburg, Pa.  
 Burleigh, W. Forrest, Oriskany Falls, N. Y.  
 Caley, D. R., Fisherville, Ont.  
 Canuel, J. L., Mont Joli, Que.  
 Chasmar, Raymond G., Hanley, Sask.  
 Childs, Thomas, Killam, Alta.  
 Christy, C. M., Brookville, Pa.  
 Claffey, T. J., Oconomowoc, Wis.  
 Clark, Judson H., Pontiac, Mich.  
 Clarkson, Wm. M., Toronto, Ont.  
 Cleaves, L. Sherman, Bar Harbor, Me.  
 Cleveland, H. R., Danville, Que.  
 Cobbett, Norman G., Helena, Mont.  
 Cockerton, Geo. C., Toronto, Ont.  
 Colby, Stanley G., Plymouth, Mich.  
 Colgan, Louis C., Tyndall, S. Dak.  
 Cooling, Thomas F., Francis, Sask.  
 Connolly, John J., Indiana, Pa.  
 Cornehl, Hugo, Detroit, Mich.  
 Coulter, I. P., Athens, Ga.  
 Covington, N. G., Auburn, Ala.  
 Craft, J. S., Des Moines, Iowa.  
 Curtis, W. L., Los Angeles, Calif.  
 Cushing, Edward R., Ithaca, N. Y.  
 Daigneault, Frederic A., Montreal, Que.  
 Delaquais, L., Notre Dame de Lourdes, Man.  
 Deming, David F., Massena, N. Y.  
 De Moulin, D. E., Lancaster, Ont.  
 Devereux, R. W., Brantford, Ont.  
 Donham, C. R., Corvallis, Ore.  
 Dorning, Joseph V., Catawissa, Pa.  
 Dunn, John, Barrie, Ont.  
 Dupre, G. J. A., Ste. Anne de la Pocatiere, Que.  
 Dwyer, Carl W., East Berkshire, Vt.  
 Egan, Francis D., Detroit, Mich.  
 Eggleston, Harry W., Alden, N. Y.  
 Ellett, Wm. H., Midlothian, Va.  
 Allis, Chas. H., Barlow, Ky.  
 Fincher, Myron G., Ithaca, N. Y.  
 Fish, Jas. G., Jr., Jacksonville, Fla.  
 Fortney, R. J., Willard, Ohio.  
 Fulton, John S., Saskatoon, Sask.  
 Gallagher, Wm. J., Yetter, Iowa  
 Gazi, Victor R., Cairo, Egypt.  
 Gingery, Howard L., Crawfordsville, Iowa.  
 Given, R. A., Reno, Nev.  
 Gladfelter, Oscar E., York, Pa.  
 Gleason, John L., Toronto, Ont.  
 Gregory, Emory I., Whitney Point, N. Y.  
 Gunn, Wallace R., Finch, Ont.  
 Gwatkin, Ronald, Guelph, Ont.  
 Haner, Frank H., Hensonville, N. Y.  
 Hansen, Frank K., Marquette, Mich.  
 Harris, C. S., Magog, Que.  
 Hastings, J. Walter, Cambridge, Md.  
 Hatchett, Guy F., Winchester, Tenn.  
 Haxby, John W., Clarinda, Iowa.  
 Heater, Verne A., Fort Dodge, Iowa.  
 Heath, Lionel M., Hull, Que.  
 Heatley, Thos. G., Woodbridge, Suffolk, England.  
 Hills, John V., Gowanda, N. Y.  
 Hodges, Harry G., Owego, N. Y.  
 Johnson, Samuel R., Lansing, Mich.  
 Karara, H. Shaker, Tanta, Egypt.  
 Kay, Arthur J., Frankfort, Ky.  
 Kennedy, Bruce D., Ottawa, Ont.  
 Kennedy, Kimball M., Waterbury, Vt.  
 Krowl, Chas. J., Corning, N. Y.  
 La Fond, Louis H., Flint, Mich.  
 Landis, Paul J., Weyers Cave, Va.  
 Lane, Theodore F., Ann Arbor, Mich.  
 Lange, Fred W., Aberdeen, S. Dak.  
 Lawrence, Geo. C., Lindsay, Ont.  
 Legault, E. C., L'Epiphanie, Que.  
 Leighton, Raymond H., Rochester, N. H.  
 Levie, T. M., Athens, Ga.  
 Little, Geo. W., New York, N. Y.  
 Littlehales, John E., Regina, Sask.

- Long, Wm. M., Baldwinsville, N. Y.  
 Loomis, Frank J., Watertown, N. Y.  
 McAlpine, D., Brockville, Ont.  
 McFarland, Austin M., Regina, Sask.  
 McGarth, Wm. E., Chicago, Ill.  
 McGhee, J. F., Chamberlain, S. Dak.  
 McIlmurray, Morgan, Caseville, Mich.  
 McIntosh, R. A., Guelph, Ont.  
 McIntyre, Alex. T., Brown City, Mich.  
 McLean, B. Courtney, Erie, Pa.  
 Mabey, M. H., Cuba, N. Y.  
 Mancill, D. Duer, Kennett Square, Pa.  
 Marquardt, Emmett H., Atlanta, Ill.  
 Martin, Walter, Jonesboro, Ark.  
 Melanson, James T., Montreal, Que.  
 Merriman, Robt. W., Auburn, Ill.  
 Metzger, Herbert J., Ithaca, N. Y.  
 Meyers, L. D., Denver, Colo.  
 Miller, Chas. L., Georgetown, Texas.  
 Miller, G. G., Council Bluffs, Iowa.  
 Monroe, Roswell M., Bainbridge, N. Y.  
 Morgan, Wm. J., Kingston, Ont.  
 Muecke, Frank J., Indianapolis, Ind.  
 Meyers, Walter L., Holly, Colo.  
 Neary, W. E., Boise, Idaho.  
 Nicholls, Wm. E., Winnipeg, Man.  
 Niehaus, Herman R., Holyoke, Colo.  
 Noffsinger, C. H., De Queen, Ark.  
 Olson, Clark W., So. St. Paul, Minn.  
 O'Neill, James J., Roberval, Que.  
 Osborne, Howard D., Austin, Minn.  
 Osman, Sayyed M., Sakha, Egypt.  
 Page, Fred W., Danielson, Conn.  
 Parks, C. S., Albemarle, N. C.  
 Patterson, Fred D., Jr., Auburn, Ala.  
 Paul, Haven T., Portsmouth, N. H.  
 Pavia E., Carlos, Mexico, D. F. Mexico  
 Phillips, Joseph C., Edmonton, Alta.  
 Piché, Marcelin A., Montreal, Que.  
 Pollard, C. M., Jacksonville, Fla.  
 Pook, Geo. G., Calgary, Ala.  
 Porter, Earl W., Reynoldsburg, Ohio  
 Ponder, Chester A., Poplar Bluff, Mo.  
 Pratt, Leonard R., Aurora, Colo.  
 Redfield, Knowlton T., Villamont, Va.  
 Richards, H., Indian Head, Sask.  
 Romberger, Earl E., Reading, Pa.  
 Ronsse, August J., Turlock, Calif.  
 Rueter, Geo. W., Berthoud, Colo.  
 Ryan, C. L., Dexter, Me.  
 Sadek, Ahmed, Ebshan, Egypt.  
 Schaefer, John A., Bangor, Mich.  
 Schilt, C. C., Edmonton, Alta.  
 Schneider, C. P., Sioux Falls, S. Dak.  
 Scott, Harry P., Fort Morgan, Colo.  
 Senécal, Lucian, Calgary, Alta.  
 Shannon, Russell S., Fort Robinson, Nebr.  
 Sheffield, Earle F., Detroit, Mich.  
 Smith, Earl D., Plainwell, Mich.  
 Smith, Hubert C., Ames, Iowa  
 Snyder, Llewellyn N., Dunnville, Ont.  
 Spaulding, Roy H., White Plains, N. Y.  
 Spearman, Wilbert, Ottawa, Ont.  
 Staggs, Geo. W., Chehalis, Wash.  
 Stiles, Floyd E., Battle Creek, Mich.  
 Strange, Chas. R., Johnstown, Colo.  
 Stuart, John McL., Ottawa, Ont.  
 Swenerton, Lorne D., Vancouver, B. C.  
 Taylor, Chas. E., East Angus, Que.  
 Temimy, Hussein El, Zagazia, Egypt.  
 Tenney, Norman H., White River Junction, Vt.  
 Theoret, J. Adelard, Montreal, Que.  
 Thorndike, Geo. M., Alto, Mich.  
 Tiffany, Arthur L., Monroe, Mich.  
 Turgeon, Elie, Quebec, Que.  
 Veilleux, J. M., St. Georges East, Que.  
 Walters, Percy K., Thorndale, Ont.  
 Way, A. J., Edmonton, Alta.  
 Whitmore, Matthew F., Addison, N. Y.  
 Wiest, Sam W., Santa Fe, N. Mex.  
 Wilson, Homer A., Jefferson City, Mo.  
 Wilson, John H., London, Ont.  
 Wolf, Chas. F., Eau Claire, Mich.  
 Yabsley, Francis C., Cissna Park, Ill.  
 Yarborough, J. H., Lowryville, S. C.  
 Younie, A. R., Omeme, Ont.  
 Zurkow, Morris L., Dover, Del.

### EARNED THEIR OATS

Some may be interested in knowing that the famous French runner, Ksar, was the greatest turf winner during 1923, having earned \$320,000 for his owner.

Isinglass, the English horse, was second with \$291,275. Donovan, another British Thoroughbred, was third with \$277,215, while the American stepper, Zev, was fourth with \$257,936.

## OTHER MEETINGS

### IDAHO VETERINARY MEDICAL ASSOCIATION

The Idaho Veterinary Medical Association met in conference and held a clinic, at the hospital of Dr. J. C. Sorensen, Idaho Falls, Idaho, August, 20-21, 1923. Veterinarians to the number of 25 were present from Idaho, Montana, Utah and Washington.

Hon. Ralph A. Lewis, Mayor of Idaho Falls, very cordially welcomed the veterinarians, and in the absence of the President, Dr. Ray B. Hurd, the response was made by Dr. J. E. McCoy, Vice-President, of Twin Falls, Idaho.

Dr. Hadleigh Marsh, of Helena, Mont., read a splendid paper on "The Bacteriology of Progressive Pneumonia of Sheep," published in the JOURNAL OF THE A. V. M. A., December, 1923.

Dr. H. E. McMillan, Filer, Idaho, presented a paper, "The Veterinarian's Relation to Public Health." Dr. McMillan ably presented his ideas of the interest and activities which veterinarians should take in matters of public health in their communities.

Dr. E. E. Wegner, Dean of the College of Veterinary Medicine, Washington State College, gave a very interesting and instructive address. The afternoon session consisted of clinics on avian tuberculosis, castrating, caponizing and demonstrations in hog cholera immunization. These clinics were participated in by all veterinarians present.

On the morning of the 21st, a clinic was held at the dairy ranch of Mr. Carl Johnson, near Idaho Falls. Practical demonstrations in the treatment of abortion disease and sterility in cattle were ably conducted under the direction of Dr. J. E. McCoy. Mr. Carl Johnson discussed animal husbandry, directing his talk towards dairy types and breeds.

A sumptuous dairy lunch was served at Mr. Gustafson's garden, a beautiful dairy ranch home near Mr. Johnson's ranch. Mr. Gustafson is generally noted for his hospitality. The beautiful surroundings and his congenial spirit, together with the splendid service rendered by the attractive young ladies, made our luncheon a very pleasant one indeed.

In the afternoon the clinics were continued at the Carl Johnson ranch and consisted of equine and bovine surgery, under the

direction of Dr. J. C. Sorensen, of Idaho Falls, and Dr. W. H. Lee, of Salmon, Idaho.

It was unanimously agreed by those in attendance that this meeting was one of the most instructive and profitable for the practitioners of any meeting in the history of the Association. At the closing session, resolutions of appreciation were adopted for the outside veterinarians who contributed to the success of the meeting, and a vote of thanks was given Dr. Sorensen for his efforts in securing material for the clinic and his hospitality shown to all veterinarians while attending the meeting in his home city.

J. D. ADAMS, *Secretary*.

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#### VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was called to order by the President, Dr. J. Elliott Crawford, in the Academy of Medicine, 17 W. 43rd St., on Wednesday evening, February 6, 1924. The minutes of the January meeting were read and approved.

Dr. O. E. McKim, of the Laboratories of Dr. Fenton B. Turck, New York City, read a paper on "Tissue Extract as an Etiological Factor in Disease." This paper was a very scientific and carefully prepared article. Drs. W. Reid Blair, Slawson and Carraba discussed it.

Dr. Chas. S. Chase read a most practical and interesting paper on "Common Diseases of the Rabbit and Their Treatment." Dr. Chase gave a very lucid description of a number of these diseases prevalent in the country and gave very practical ideas of how to treat them; also how many of them can be prevented by proper disinfection. The paper was discussed by Drs. W. Reid Blair, Clayton, McKinney and others.

Dr. Robert S. MacKellar exhibited a specimen of a very large polypus removed from the nostril of a horse. This specimen was unusually large but was removed with comparative ease.

Dr. J. W. McKinney reported good results in treating three cases of purpura in horses recently. Dr. Amling reported good results in treating purpura and advised plenty of fresh air and exercise, when possible, with pasture. Dr. C. E. Clayton reported a number of interesting cases, including a dislocated or fractured cervical vertebra.

Dr. R. W. Gannett gave a splendid report of the Conference of Veterinarians, at Ithaca. He reported a large attendance and a goodly number of instructive papers read.

Dr. C. N. Darke was elected to membership in the Association. A vote of thanks was extended Drs. McKim and Chase for their contributions to the program.

No further business appearing, the meeting adjourned.

#### MARCH MEETING

The regular monthly meeting of the Veterinary Medical Association of New York City was called to order by the President, Dr. J. Elliott Crawford, in the Academy of Medicine, 17 West 43rd St., on Wednesday evening, March 6, 1924. The minutes of the February meeting were read and approved.

Dr. C. H. Higgins, of the Lederle Laboratories, Pearl River, N. Y., addressed the meeting on the relationship of human medicine, veterinary medicine and veterinary education. He pointed out that veterinary medicine has been a great factor in human medicine, with which it has always been closely allied.

The veterinary students, years ago, were allowed to spend several years with the medical students, in many colleges, and this association and the surroundings of the medical colleges gave the student a splendid start. It was Dr. Higgins' belief that in the future there will be a closer correlation between the medical and veterinary students, made necessary on account of the similarity of parasitology. He referred to the valuable work done by Prof. Theobald Smith, in the discovery of the parasite causing Texas fever, and to the great good done to the country.

Dr. Higgins said the present trend in medicine was toward laboratory work, but while this is of great importance it can be overdone, in delaying treatment. Referring to the student, he advised an apprenticeship, but care should be taken to choose a man who sees the human side of the profession. In practice he should identify himself with the community, politically and otherwise. He believes the schools show the lack of a business course, as young men many times do not know how to meet the public properly.

He referred to diet and the elements entering into it as a common cause of bad teeth. Peaches and oranges are an important factor to the well-being of the digestive tract. Molasses feeds have been a factor in reducing the number of colics in horses. The intravenous methods of treatment are good in

certain instances but should not be used too much. The veterinarian has a much wider field than the medical man, due to the enormous quantity of food supplies he must supervise.

A rising vote of thanks was extended Dr. Higgins for this practical and instructive talk. Dr. Geo. H. Berns, in discussing Dr. Higgins' remarks, spoke of early veterinary education and believed the medical and veterinary schools should be combined.

Dr. W. Reid Blair spoke of the question of diet, in the New York Zoological Park, where most of the animals have been living an artificial life. He has been able to benefit many animals by using calcium, phosphates, iodids, Epsom salts, etc., in compounds. Drs. C. W. Shaw, J. Payne Lowe and others exchanged ideas on the subjects.

Dr. Geo. W. Little read a carefully prepared paper on "Feeds and Feeding of Small Animals." Discussion on this paper was deferred until the next meeting owing to the lateness of the hour.

Dr. Alfred T. Baeszler, of Staten Island, Dr. John Halloran, of Staten Island, Dr. J. Payne Lowe, of Passaic, N. J., and Dr. J. E. Robbins, of Bayshore, N. Y., were elected to membership in the Association.

It was regularly moved and seconded that the sum of \$100 be transferred from the prosecuting fund to the general fund. Announcement of the alumni meeting and dinner to be held at Cavanagh's, 258 W. 23rd St., New York, March 11, was made.

No further business appearing, the meeting adjourned.

C. G. ROHRER, *Secretary*.

### MICHIGAN-OHIO VETERINARY ASSOCIATION

The regular annual meeting of the Michigan-Ohio Veterinary Association was held at Adrian, Mich., April 16, 1924, with about 25 members in attendance. The following program was presented:

"Disinfecting and Disinfectants," by Prof. W. L. Mallman, M. A. C., East Lansing, Mich.

"Dog Distemper and Its Complications," by Dr. E. K. Sales, M. A. C., East Lansing, Mich.

"Bacteriology. What is It?" by Dr. Ward Giltner, M. A. C., East Lansing, Mich.

"Pitfalls in Practice," by Dr. Judson Black, Lansing, Mich.

"Forage Poisoning," by Dr. B. J. Killham, Lansing, Mich.

In each case a very interesting discussion followed. Particularly was this true of the subjects presented by Dr. Sales and Dean Giltner. In the discussion of canine distemper much was said pro and con as to the value of the various biological products being used in the prevention and treatment of this disease. There was a very animated discussion of the merits and demerits of the serological tests used in connection with infectious abortion.

A motion prevailed that the Association endorse the report of the Committee on Policy of the A. V. M. A., as presented at the 1923 meeting, at Montreal, and subsequently published in the JOURNAL OF THE A. V. M. A. This motion was carried unanimously.

Election of officers for the ensuing year resulted as follows: President, Dr. J. H. Lenfesty, Lyons, Ohio; Vice-President, Dr. C. E. Moorman, Tecumseh, Mich.; Secretary-Treasurer, Dr. W. E. Watson, Metamora, Ohio.

W. E. WATSON, *Secretary.*

#### K. S. A. C. VETERINARY MEDICAL SOCIETY

The annual banquet of the K. S. A. C. Veterinary Medical Society was held at the Gillette Hotel, Manhattan, Kansas, the evening of April 17, 1924. Mr. E. E. Hodgson acted as Toastmaster, and called upon the following speakers: Dr. H. C. Gale, Clyde, Kansas, President of the Kansas Veterinary Medical Association; Major R. J. Foster, Ft. Riley, Kansas; Dean F. D. Farrell, of the Division of Agriculture, K. S. A. C.; President W. M. Jardine, K. S. A. C.; Dr. O. O. Wolf, Ottawa, Kansas, President of Kansas Board of Veterinary Medical Examiners; and Dr. R. R. Dykstra, Dean of Division of Veterinary Medicine, K. S. A. C.

The following elections and prizes in veterinary medicine were announced:

*Kansas State Agricultural College, Honor Society of Phi Kappa Phi*

Edward Raymond Frank, of Manhattan, Kansas.

William Taylor Miller, of Los Angeles, California.

*Kansas State Agricultural College, Honor Society of Agriculture, Gamma Sigma Delta:*

William Taylor Miller, of Los Angeles, California.

Charles James Coon, of Manhattan, Kansas.

Ernest Eugene Hodgson, of Harveyville, Kansas.

#### FACULTY PRIZE

A prize of \$25.00, donated annually by the Veterinary faculty, to the student making the highest average grade in the veterinary curriculum during his four years of attendance:

Charles James Boon, of Manhattan, Kansas.

#### KINSLEY PRIZE IN PATHOLOGY

An annual prize of \$25.00, donated by Dr. A. T. Kinsley, ex-President of the American Veterinary Medical Association, Kansas City, Missouri, to the student making the highest average grade in veterinary pathology:

Edward Raymond Frank, of Manhattan, Kansas.

#### SCHMOKER PRIZE IN CLINICS

A prize of \$25.00, donated annually by Dr. Edw. A. Schmoker (K. S. A. C. '17), veterinarian for the Carnation Stock Farms, Tolt, Washington, to the student making the best grade in veterinary clinics:

Ernest Eugene Hodgson, of Harveyville, Kansas.

#### GINGERY PRIZE IN SURGERY

An annual prize of \$25.00, donated by Dr. J. B. Gingery (K. S. A. C. '10), veterinary practitioner, Muscatine, Iowa, to the student obtaining the highest grades in veterinary surgery:

Edward Raymond Frank, of Manhattan, Kansas.

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### TRI-COUNTY VETERINARY ASSOCIATION

The annual meeting of the Tri-County Veterinary Association was held at the Court House, Richmond, Ind., April 25, 1924. The principal speakers on the program were Dr. Edward A. Cahill, Director, Biological Laboratories, Pittman-Moore Co., Indianapolis, and Dr. R. A. Whiting, Veterinary Department, Purdue University, LaFayette. Tuberculosis eradication and poultry diseases were the topics presented.

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### NORTHEASTERN INDIANA VETERINARY MEDICAL ASSOCIATION

The monthly meeting of the Northeastern Indiana Veterinary Medical Association was held at the Chamber of Commerce, Fort Wayne, Ind., April 22, 1924. The members attended the Fort Wayne Dog Show in the afternoon. In the evening the members and their wives partook of a banquet. At a business

session it was decided to hold the June meeting at Tri-Lake, June 16. Dr. W. G. Clark, of Columbia City, Ind., and Dr. L. A. Merillat, of Chicago, Ill., were the principal speakers on the program.

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### NATIONAL ASSOCIATION OF B.A.I. VETERINARIANS MISSISSIPPI VALLEY DIVISION

The regular monthly meeting was held at St. Louis, Mo., Saturday, May 3, in the Federal Building. Meeting called to order by Chairman Dr. R. C. Lambert. Minutes of the previous meeting read and accepted.

► By special request, Dr. M. L. Crans again read his paper on "Tuberculosis," which was followed by a discussion by Drs. Pease, Jenison, Lambert, Crans, Maloney, Bruns, Thurmon and Surring.

Following this, the subject of "Cervical Abscesses" was discussed by Drs. Lambert, Pease, Thurmon and Jenison.

Dr. Jenison made a report on the status of foot-and-mouth disease eradication in California. Dr. L. C. Stewart sent some photographs of the foot-and-mouth campaign in California which were appreciated by the members present.

The following members were present: Drs. H. H. Brown, G. H. Bruns, M. L. Crans, J. S. Jenison, R. C. Lambert, A. J. Maloney, J. F. Pease, R. J. Spain, R. E. Surring, F. S. Thurmon and H. J. Timmerman.

G. H. BRUNS, *Secretary*.

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### NEWS ITEMS

There are thirty-two life members on the roll of the Pennsylvania State Veterinary Medical Association. Life membership is conferred upon the completion of twenty-five years of continuous membership. Life members have all the privileges of active members, but pay no dues.

Milk producers of Cedar Rapids, Waterloo, and Dubuque, Iowa, are formulating plans for a "Drink More Milk" campaign, to extend over a period of about one year. The expense will be borne by the producers, who will contribute to the fund at the rate of one cent per one hundred pounds of milk distributed.

A rather serious outbreak of rabies recently occurred in Davis and Livingston Counties, Missouri. Quite a number of cattle and hogs have been lost, following bites by rabid dogs.

## **ARMY VETERINARY SERVICE**

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### **THE 65TH CAVALRY DIVISION WANTS VETERINARY OFFICERS**

The Officer's Reserve Corps in this Corps Area (Illinois, Michigan, and Wisconsin) has vacancies for veterinarians, particularly in the lower grades. The 65th Cavalry Division, Organized Reserves, with headquarters in Chicago and units throughout the three states, has vacancies that it would like to fill. Anyone interested should correspond with Major Robert C. Musser, Veterinarian, Sixth Corps Area, 1819 W. Pershing Road, Chicago, Illinois, who will place requests for information and blank forms for application for commissions in the Officers Reserve Corps in the proper hands.

It is probable that there is a lack of reserve veterinary officers in other Corps Areas. If you are not in the Officers Reserve Corps, you should join immediately. If you are not in the 6th Corps Area, write Lt. Col. J. A. McKinnon, Director, Veterinary Corps, Surgeon General's Office, Washington, D. C., for information.

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### **TRAINING OF OFFICERS IN THE BRANCH ASSIGNMENT GROUP OF THE MEDICAL DEPARTMENT RESERVE**

It is proposed to hold training camps for officers of the Medical Department Reserve at the following camps, for a period of two weeks, beginning about July 7, 1924:

At Carlisle Barracks, for the 1st, 2d, 3d, 4th, and 5th Corps Areas.

At Fort Snelling, for the 5th and 7th Corps Areas.

At Camp not designated, for the 8th Corps Area.

At Camp not designated, for the 9th Corps Area.

Funds will be allotted for the training of officers of the Medical Department Reserve assigned to the *Branch Assignment Group* under the direction of the Surgeon General. It is planned to give officers of the various sections of the Medical Department Reserve Corps, who can accept training during this period, the advantage of full field instruction, including tactics and technique of operation of divisional medical units. During the training

period, troops will be available to demonstrate the general and selective operation of these units, and it is proposed to make the training intimate and practical, presenting to the Reserve officer the opportunity to exercise command and put into execution plans simulating the actual conditions which will present when they are called upon in emergency.

It is hoped that as many officers as possible, for which funds are allotted, will take advantage of the splendid opportunity which this camp affords for training and for indication of their support of the medical program.

Officers of the *Branch Assignment Group*, who indicate their desire to accept training during the period mentioned, will, when ordered to active duty, receive mileage from their home to camps and return, and the pay and allowances of their grade while on active duty.

It is requested that those desiring to receive this training will complete the form below, which should be mailed to The Surgeon General of the Army:

Name:.....

Rank:.....

Address:.....

I do \_\_\_\_\_ request that I be ordered to active duty for the  
(or "not")  
training period, about July 7-21, 1924. I have \_\_\_\_\_ been on  
(or "not")  
active duty for training since June 30, 1924.

Signature:.....

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### NEWS ITEMS

An outbreak of rabies was recently reported at Washington, Ind., by Dr. Omar Fleener.

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Hog raisers of Hillsdale County (Mich.) have shipped 6185 hogs during the past two months, on which they have received the ten-cent premium paid by packers on hogs coming from modified accredited areas. Hillsdale County was placed in this classification, November 15, 1923. Over \$1200 in premiums has been returned to the County in this way.

## COMMENCEMENT

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### ONTARIO VETERINARY COLLEGE

A special convocation was held in Convocation Hall, University of Toronto, May 1, for the purpose of conferring the degree of Bachelor of Veterinary Science upon the 26 students graduated this year. The graduates of the School of Practical Science also received their degrees upon the same occasion. Promptly at 4 o'clock the students, academically gowned, filed in and took the seats allotted to them. Shortly afterwards the Chancellor of the University, together with President Falconer, the Dean of the School of Practical Science, the Principal of the Ontario Veterinary College, and the members of the Senate of the University marched in and took their places.

This occasion being the first appearance of Sir William Mulock in his newly appointed position as Chancellor of the University, the Dean of the S. P. S., on rising to confirm the graduation of his students, first tendered his congratulations to his honor on his appointment. Dr. C. D. McGilvray, Principal of the Ontario Veterinary College, then arose and after tendering his respects to the Chancellor certified to the graduation of the veterinary students, and they filed up to the seat of the Chancellor and had the degree of Bachelor of Veterinary Science conferred upon them.

At the conclusion of the ceremonies the Chancellor gave an address and with a few well chosen words congratulated the students and paid tribute to the Ontario Veterinary College and its Faculty for the distinction it must have gained as an educational institution in that branch of science since he had noted that students were in attendance from European countries, from the British West Indies, and also from many of the States of the great sister country to the south of us. He also stated that it was gratifying to know that most of the graduates had places to go, that they were quickly taken up and absorbed to fulfill their life's work and ambitions, that it spoke well for the usefulness of their profession and the condition of affairs in the country at large. Following the Chancellor, Sir Robert Falconer, President of the University, briefly addressed the audience, and announced a reception by the Chancellor, himself and Lady

Falconer, for a short social intercourse, and at which a light lunch was served.

Degrees were conferred on the following: J. G. Anderson, W. M. Bickell, G. R. Booth, E. R. Carpenter, A. F. Davis, D. N. English, A. E. C. Goodman, V. A. Grant, J. S. Gray, C. E. Hagyard, E. E. I. Hancock, R. L. Hanley, P. E. Home-Hay, C. H. Holmes, R. M. Jones, C. Latimer, H. S. C. MacDonald, S. R. McKelvey, L. D. Perry, H. A. Pugh, C. R. Quillin, C. R. Quinlan, B. J. Riley, J. E. Shirley, H. P. Westerberg, W. B. White.

Honors were awarded as follows:

*General Proficiency*

First prize—H. P. Westerberg, of Meadowvale, Ont.

Second prize—John S. Gray, of Newton, N. J.

Third prize—E. E. I. Hancock, of Port Hope, Ont.

*Bacteriology (Awarded by Dr. R. Gwatkin)*

C. E. Hagyard, of Lexington, Ky.

*The Helen Duncan McGilvray Honorarium*

A. E. C. Goodman, of Toronto, Ont.

*Canadian Army Veterinary Corps Prize*

G. R. Booth, of Edwards, Ont.

R. A. McL.

## THE VETERINARIAN

The conscientious and efficient veterinarian, like the old family doctor, is an individual whose services are appreciated only in case of need, and we feel that it is a grave mistake to pass legislation, as has been done in several states, intended to hamper his usefulness. While the profession has not been free of fakirs and grafters, yet we are not willing to believe it is burdened with this element more than other occupations. No line of business or profession is entirely free from selfish men, those who stoop to disreputable tactics and practices. We personally know many veterinarians and are mighty proud to think we have several warm friends among them. As a class the veterinarians are worthy of respect and confidence, and the live stock industry would suffer a great loss if they should be driven into other lines of work. Yet that is just what will happen if this spirit of hostility, expressed through the laws of various state legislatures, is permitted to go on unchecked.—(Editorial in *The Spotted Poland China Breeder*, May 1924).

## MISCELLANEOUS

### EASTERN STATES CONFERENCE AT ALBANY

An excellent program has been arranged for the joint meeting of the Eastern States Conference on Tuberculosis Eradication and the New York State Veterinary Medical Society, at Albany, N. Y., June 10-11-12, 1924, as announced in the May issue of the JOURNAL. Sessions will be held in Chancellors' Hall, State Education Building. Headquarters will be at the Ten Eyck Hotel.

The meeting will cover three days. Tuesday and Wednesday, June 10-11, will be given over to the deliberations of the Tuberculosis Eradication Conference, the program of which has been arranged by Dr. J. A. Kiernan. Hon. Berne A. Pyrke, Commissioner of Farms and Markets, Albany, N. Y., will deliver the address of welcome and Hon. A. L. Felker, Commissioner of Agriculture, Concord, N. H., will make the response. Dr. John R. Mohler will deliver the key-note speech, outlining the object of the Conference.

Reports of progress in the various states will be made by the state sanitary control officials, completing the morning program. In the afternoon papers will be presented, bearing on special phases of the work. Tuesday evening there will be a moving picture entertainment. The program will be continued Wednesday morning and afternoon, with a number of nationally known speakers, including Commissioner H. R. Smith, of Chicago, Dean V. A. Moore, Mr. A. J. Glover, Editor of Hoard's Dairyman, Dr. C. H. Stange, President of the A. V. M. A., Senator Copeland, of New York, Dr. J. G. Ferneyhough, Dr. E. C. Schroeder, Dr. H. S. Beebe, Hon. Nathan Straus, Jr., Dr. James G. Townsend, U. S. Public Health Service, and Hon. C. F. Bigler, President of the N. Y. State Holstein-Friesian Association.

For Thursday, June 12, Secretary Hayden has provided a very attractive program, in behalf of the N. Y. State Veterinary Medical Society. Papers will be presented by Drs. J. N. Frost, W. A. Young, F. E. McClelland, George W. Little, H. J. Milks and Prof. J. D. Brew, all on subjects of considerable interest to the practitioner. A banquet will be served, at the Ten Eyck Hotel, probably Wednesday evening.

### SERUM COMPANIES FIGHT IT OUT

Some time ago the Waterloo Serum Laboratories, of Waterloo, Iowa, entered suit against the United Serum Company, of Kansas City, Kansas, to recover damages to the extent of \$40,000. Suit was brought on three counts: alleged violation of contract, negligence, and fraudulent representation. Thirty thousand dollars was asked for alleged loss of plaintiff's business in Northeastern Iowa, and \$10,000 for alleged loss of profits during 1920.

The defendant filed a demurrer, denying the allegations, and claimed that the only guarantee in the contract was that the serum and virus were to be produced according to accepted methods, to be free from harmful substances, and made in accordance with the regulations of the Federal Department of Agriculture.

Judge E. B. Stiles, of the Iowa District Court, sustained the first and third counts, but overruled the second, which alleged negligence on the part of the defendant in furnishing anti-hog cholera serum and virus to the plaintiff. Judge Stiles said: "Inasmuch as the Court is of the opinion that count two sets forth a good cause for action, defendant's demurrer to the petition as a whole, is overruled."

The following is taken from the ruling by Judge Stiles:

"While it is doubtless true that, in a case such as is presented at bar, the degree of care required of the manufacturer is not so high as that which is required of manufacturers of products intended for human food, yet it is certainly true that a manufacturer of remedies for hogs, which may easily become highly dangerous to hog life and readily produce disease quickly fatal to any hog to which the alleged remedy is applied, owes a duty to persons purchasing this product. This duty involves the use of a high degree of care in the preparation and preservation of the products.

This Court is of the opinion that count two (negligence in preparation) of the petition presents a good cause of action. See *Davis vs. Van Camp Packing Company*, 189 Iowa 775, etc."

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### REEL BUSY

"I believe," said the impatient veterinarian as he put aside the telephone, "that I'll go fishing."

"Didn't know you cared for fishing."

"I don't, ordinarily, but it's the only chance I have of finding myself at the end of a line that isn't busy."

## NECROLOGY

### HERVEY T. POTTER

Dr. H. T. Potter, inspector-in-charge, Bureau of Animal Industry, Calais, Maine, died April 16, 1924. He was stricken with bronchitis, while on an inspection trip, among the lumber camps of Northern Maine, and was able to return home after a few days. He was compelled to spend a month in the hospital, however, and when apparently on the road to recovery, returned to his office to resume his official duties. He suffered a relapse, and died of infection of the sinuses, complicated with erysipelas.

Born at New Haven, Conn., September 22, 1857, of pioneer New England stock, he displayed an early love for animals. He first took up blacksmithing. Later, after he had assumed the responsibilities of a family, he decided to study veterinary medicine, and entered the New York College of Veterinary Surgeons, being graduated in 1892. For a number of years he was a member of the teaching staff of his Alma Mater.

In 1898, Dr. Potter moved to Calais, Me., and shortly thereafter entered the service of the Bureau of Animal Industry, on a per diem basis, in connection with the inspection of import and export animals, crossing between Canada and the United States. This work developed to such a degree that Calais was made a station of the B. A. I., with an office and field force, Dr. Potter in charge. He was detailed to Pennsylvania, on the foot-and-mouth disease outbreak of 1908.

In all his work, Dr. Potter always gave the best that was in him. With never the slightest inclination to do things in a spectacular way, he went about his work in a way that invariably brought results. Dr. Potter represented the type of veterinarian, imbued with loyal devotion to his work, that has made the B. A. I. the greatest organization of its kind.

Dr. Potter joined the A. V. M. A. in 1918, and was in attendance at the Montreal meeting, in August last. He is survived by his widow, one daughter and one son, Dr. Geo. M. Potter, now practicing in Portland, Me.

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### FRANK E. GLEZEN

Dr. Frank E. Glezen, a registered non-graduate practitioner, of Lisle, N. Y., died suddenly, April 4, 1924.

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**MICHAEL P. FIEDLER**

Dr. M. P. Fiedler died suddenly at his home in Millheim, Pa., February 15, 1924.

Born on a farm, near Woodward, Pa., May 4, 1883, he received his early education in the township schools. He attended Central Pennsylvania College, at New Berlin, 1901-1902. He entered the University of Pennsylvania in 1909, and received his veterinary degree in 1912. While at Pennsylvania, Dr. Fiedler was prominent in athletics, and was regarded as an almost perfect specimen of physical development. In strength tests made in the U. of P. Gymnasium, he made a record that stood for a number of years. He also excelled in sprinting and putting the shot.

Dr. Fiedler was engaged in private practice and regarded as a highly successfully practitioner by his hosts of clients. He is survived by his father and three brothers.

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**GEORGE SELWYN JORDAN**

Dr. George S. Jordan died at his home, Williamstown, Mass., April 9, 1924, following an illness of a week, due to pneumonia.

Born at Sutton, N. H., May 26, 1880, Dr. Jordan attended the high schools of Warner and Mount Hermon, N. H. He received his veterinary degree from Ohio State University in 1909.

Dr. Jordan located in Williamstown in 1910, after spending a year in Malden, Mass. He is survived by his widow and six children.

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**FRANK E. PERKINS**

Dr. Frank E. Perkins died April 14, 1924, at his home in Ellsworth, Wis., after a lingering illness. He was in his 51st year.

Born in Red Wing, Minn., he attended high school, and later Beeman's Business College. He entered the Chicago Veterinary College, from which he was graduated in 1904, with honors. He immediately located in Ellsworth and practiced there for the past twenty years, during which period he served as a trustee of the village of Ellsworth, for several terms.

Dr. Perkins was a member of the Wisconsin Veterinary Medical Association. Also a member of the Elks, Beavers, Royal Arcanum and Knights of Pythias. He is survived by his widow, one son, one daughter, his mother, one sister and three brothers.

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**JAMES HENDRICKEN WEIBEL**

Dr. James H. Weibel, of Waldron, Ind., was found dead in bed at his home, May 2, 1924. Death was believed to be due to apoplexy. Dr. Weibel was born in Denmark, Aug. 3, 1864. He came to the United States in 1896 and took out citizenship papers in 1901.

Dr. Weibel was a graduate of the Chicago Veterinary College, class of 1909. He returned to Iowa and practiced in Keota for seven years, and later in Shelbyville, Ind., for four years. Mrs. Weibel died in 1921 and Dr. Weibel returned to Waldron, Ind., her old home, where he practiced up until the time of his death.

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**J. J. OBERST**

Dr. J. J. Oberst died at his home, Grafton, Wis., Dec. 18, 1923. He was born Mar. 21, 1867, at Belgium, Wis., and was graduated from the Chicago Veterinary College, class of 1893. Dr. Oberst practiced for a few years in Port Washington and West Bend, and in 1902 located in Grafton, Wis., where he built up a very large practice. Dr. Oberst was a member of the Wisconsin Veterinary Medical Association. He is survived by a widow and one son.

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**WILLIAM O. STILLMAN**

Dr. William O. Stillman, president of the American Humane Association, died at his home in Albany, N. Y., March 15, 1924.

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**ROBERT P. FEISER**

Dr. Robert P. Feiser, a registered non-graduate veterinary practitioner and pharmacist, of East Berlin, Pa., died recently.

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**PETER J. HIPSCHE**

Dr. Peter J. Hipschen, of Marcus, Iowa, died recently, following a protracted illness. He was a graduate of the McKillip Veterinary College, class of 1909.

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Dr. William Dimond, of Newark, N. J., died April 5, 1924. He was a graduate of the American Veterinary College, class of 1885, but had not been in active practice for some time.

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Dr. Marvin D. Stevens, of Moroni, Utah, died April 3, 1924.

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**BIRTHS**

To Dr. and Mrs. J. L. Boyle, of Lindsay, Nebr., a son, Carell James, March 27, 1924.

To Dr. and Mrs. Robert E. Warren, of Hammond, La., a son, Robert E., Jr., February 15, 1924.

To Dr. and Mrs. George A. Rathman, of Wichita, Kans., a daughter, Patricia Ann, February 24, 1924.

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**PERSONAL**

Dr. C. R. Quillin (Ont. '24) has located at Coeburn, Va.

Dr. Frank Hare (K.S.A.C. '20) is now located in Ithaca, N. Y.

Dr. C. A. Trapkey (Gr.R. '00) has located in Knightstown, Ind.

Dr. L. U. Shipley (Chi. '91) has been elected mayor of Sheldon, Iowa.

Dr. W. B. Van Cleave (Ind. '09) is County Veterinarian of Peoria County, Ill.

Dr. E. T. Hallman (Ala. P.I. '10) has been re-elected an alderman of East Lansing, Mich.

Dr. Emil Mueller (Chi. '92) was recently elected to the office of mayor of New Ulm, Minn.

Dr. T. M. Bayler (Chi. '11) gives his new address as: 315 East Chestnut St., Bloomington, Ill.

Dr. C. T. Howard (Ind. '07) is engaged in tuberculosis eradication work in St. Joseph County, Ind.

Dr. J. B. Kingery (Ind. '06), of Logansport, Ind., has disposed of his practice to Dr. V. H. Cooper.

Dr. A. J. Steiner (Iowa) has opened a small animal hospital at 184-186 Walnut Street, Lexington, Ky.

Dr. William Sheppard (R.C.V.S. '70), who removed to Florida several months ago, is now located at Orlando.

Dr. Fenner C. Smith (Corn. '18), recently of Sherman, N. Y., is now located at 311 East 6th Street, Jamestown, N. Y.

Dr. Frederick A. Hall (Corn. '23), formerly of Ithaca, N. Y., is now associated with Dr. Killips, at Oskaloosa, Iowa.

Dr. C. W. McConkey (Cinn. '14) is erecting a large modern veterinary hospital at 909 North Perry Street, Napoleon, Ohio.

Dr. N. S. Mayo (Chi. '89), of the Abbott Laboratories, has been elected president of the Export Managers Club of Chicago.

Dr. H. W. Bales (K.S.A.C. '20) has removed from Syracuse, N. Y., to Oswego, N. Y. His address is: 105 East 4th Street.

Dr. John K. Bosshart (Corn. '12), of Camden, N. Y., has been confined at his home, since January, with an attack of phlebitis.

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Dr. George C. Newberg (K. C. V. C. '06), on meat inspection work at Madison, Wis., has been transferred to Kansas City.

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Dr. Thos. T. Hartman (K. C. V. C. '09), has been transferred from National Stock Yards, to Kansas City, on meat inspection work.

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Dr. Pearl C. Fletcher (O.S.U.), of Cowden, Ill., assumed his new duties as County Veterinarian of Dewitt County (Ill.), on May 1.

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Dr. O. R. Conley (Cinn. '17), for some time stationed at Topeka, Kans., on virus-serum control, has been transferred to Indianapolis.

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Dr. F. W. Morgan (McK. '05), of Chattanooga, Tenn., lost his veterinary hospital by fire, on April 13, 1924. The loss is estimated at \$15,000.

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Dr. C. C. Dobson (Ind. '12) is credited with being the bovine tuberculosis inspector for Delaware County (Ind), according to the Muncie (Ind.) *Star*.

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Dr. Chas. H. Wright (Ont. '08), of Jackson, Tenn., recently reported the birth of a well-developed calf to a Jersey cow only thirteen months of age.

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Dr. Otto C. Anderson (McK. '15), of Detroit, recently took the Pasteur treatment for the third time in five years, following the bite of a rabid patient.

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Dr. B. M. Underhill (U.P. '95) addressed the Delhi Veterinary Club on "Parasites and their Control in Domesticated Animals," at the March meeting.

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Lt. Edward M. Curley (U.P. '11) has been detailed as Assistant Professor of Military Science and Tactics, at Cornell University, effective June 15, 1924.

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Dr. Wm. G. Chrisman (Ont. '02), Mayor of Blacksburg, Va., has accepted the appointment of City Veterinarian of Danville, Va. He assumed his duties May 1.

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Dr. Chas. W. Gates, of Baylis, Ill., has been appointed County Veterinarian by the Adams County (Ill.) Board of Supervisors, with headquarters at Quincy.

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Dr. Geo. W. Rawson (U.S.C.V.S. '16), of Detroit, Mich., was called to Richmond, Va., the latter part of April, to testify in the famous Ferneyhough-Glass case.

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Dr. Tait Butler (Ont. '05), Editor of *The Progressive Farmer*, of Memphis, Tenn., is in Europe attending the International Agricultural Institute, in Rome, Italy.

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Capt. Ralph M. Buffington (Corn. '05), for some time detailed at Cornell University, has been ordered to proceed to Fort Sam Houston, Texas, for duty, July 1, 1924.

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Dr. W. H. Feldman (Colo. '17), of Fort Collins, Colo., has been elected an honorary member of Zeta Chapter of Alpha Psi Fraternity, at the Colorado Agricultural College.

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Dr. S. D. Shoulkin (O.S.U. '20), of Allendale, S. C., Assistant State Veterinarian of South Carolina, visited the N. Y. State Veterinary College, at Ithaca, the latter part of April.

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Dr. R. S. Beaver (K.S.A.C. '23), of Shelby, Iowa, recently gave a lecture and demonstration on the control of parasitic diseases of swine, at a gathering of farmers, in Monroe, Iowa.

Dr. B. J. Killham (McK. '12), of Lansing, Mich., has been elected to honorary membership in the Iota Chapter of Alpha Psi Fraternity, at the Michigan Agricultural College.

Dr. Noble W. Elsbury (Ind. '11), of Greenfield, Ind., has had plans drawn for a modern veterinary hospital to be completely equipped for clinical laboratory and diagnostic work.

Dr. J. P. Hutton (O.S.U. '11), of the Michigan Agricultural College, was a member of the committee having in charge the M. A. C. Horse Show at East Lansing, the latter part of May.

Dr. J. I. Gibson (Ont. '87), Live Stock Commissioner of South St. Joseph, Mo., recently spent a week in Fremont County (Iowa), lecturing on animal disease control before farmer gatherings.

Dr. Stanley G. Colby (Mich. A.C. '19) has been transferred from Wayne County to Marquette County, with headquarters at the Upper Peninsula Development Bureau, at Marquette, Mich.

Dr. Emerson A. Ehmer (Wash. S.C. '18) was recently given a full-page write-up in the Sunday edition of one of the Seattle (Wash.) papers, in which his Dog and Cat Hospital was nicely featured.

Dr. W. H. Lytle (Iowa '02), State Veterinarian of Oregon, has returned from a visit to California, where he went recently to confer with the officials relative to the foot-and-mouth disease outbreak.

Dr. G. W. Hamilton (Ind. '10), of Fort Wayne, Ind., has been appointed County Veterinarian by the Noble County (Ind.) Commissioners. Dr. Hamilton began county area testing in Perry township on April 14.

Dr. G. A. Dick (U.P. '04) addressed the Central Pennsylvania Veterinary Club on "Swine Husbandry," and the Conestoga Veterinary Club on "Feeding Dairy Cattle," at the March meetings of these organizations.

Dr. W. G. Irvin (Cinn. '15), of Carlisle, Ky., for the past five years County Live Stock Inspector, has been appointed an Assistant State Veterinarian and established new headquarters at Ashland, Ky., on May 1.

Dr. A. J. DeFosset (O.S.U. '07) has been transferred from North Carolina to Ohio, in charge of Hog Cholera Control and Tuberculosis Eradication Work, with headquarters in the State House Annex, Columbus, Ohio.

Dr. Charles B. Cain (Corn. '23) has been spending the year at Iowa State College, engaged in post-graduate work in the Department of Animal Husbandry. He has been majoring in animal nutrition and minoring in meats.

Dr. B. T. Simms (Ala. P.I. '11), of Corvallis, Ore., has been spending some time in Los Angeles, Calif., as the representative of the Oregon Live Stock Sanitary Board. He is under orders to remain until the outbreak is under control.

Dr. W. A. Johnston (Chi. '11), of Taylorville, Ill., was recently the victim of a curious accident, in which he received, among other injuries, a broken right leg. Dr. Johnston's apartment was in course of being remodelled and he was on the second floor, when a truck delivering building material knocked away a prop supporting the building. Dr. Johnston came down with the crash, and had it not been for the fact that he landed beneath his veterinary operating table, on the first floor, might have lost his life beneath the weight of over 150 concrete blocks.

